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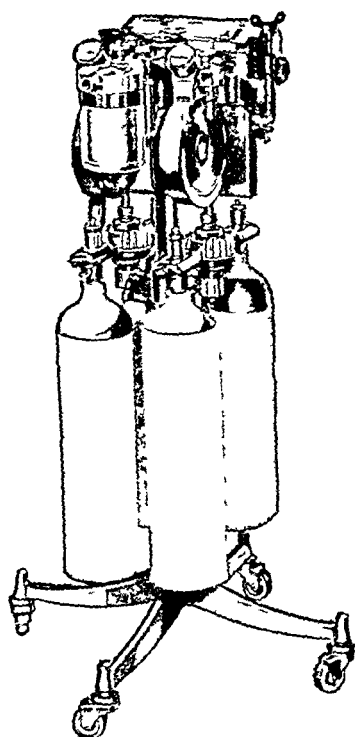
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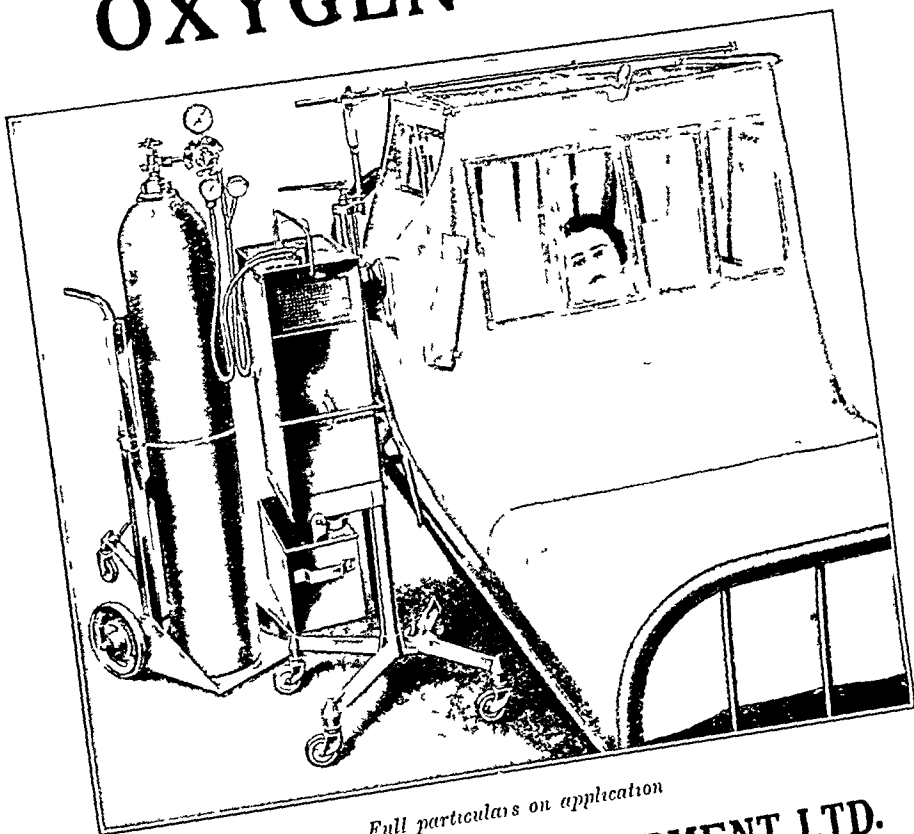
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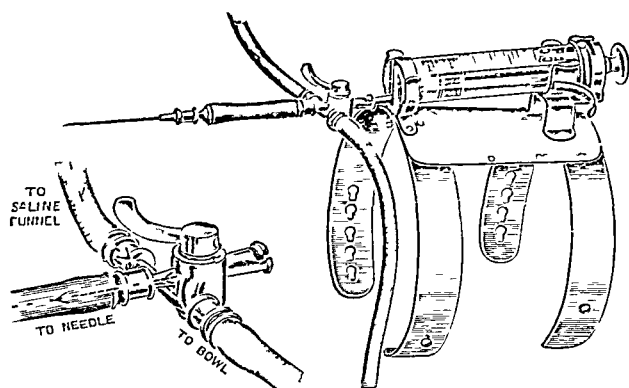
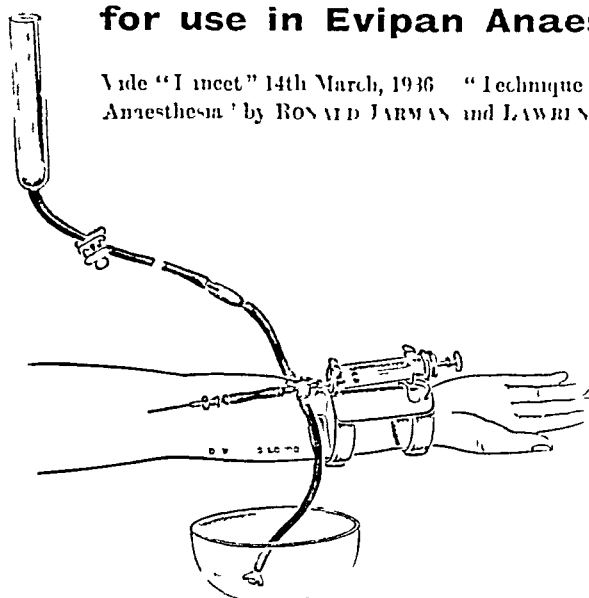
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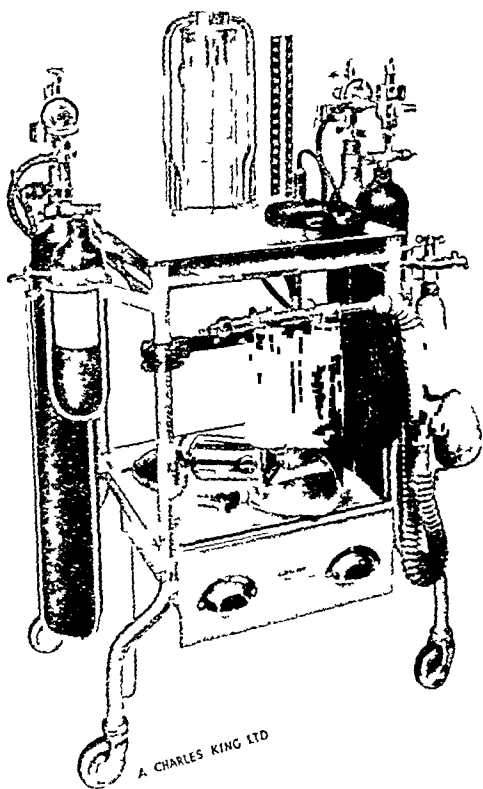
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# British Journal of Anæsthesia

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VOL XV No 2

JANUARY, 1938

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## EDITORIAL

THE difficulties which confront the teacher of anæsthetics grow ever greater. It is not simply that what has to be taught to the student in the way of practical administration of anæsthetics is more complicated and much more diverse than it was in the days of simple methods. No, his chief difficulty is to find opportunities for giving practical instruction in these complicated processes. Not only this, but the teacher knows that the average student when he is qualified and goes out into practice will not have in all probability complicated and expensive apparatus with which to work. Therefore, he ought to be made at least a safe and competent anæsthetist with the methods which will be at his disposal. In these it is still more difficult for the teacher to be able to instruct him. Such a large proportion of the operations on which the hospital anæsthetist is engaged are serious major operations that the surgeon is naturally not happy unless the anæsthetic is in the hands of the expert himself. A gastrectomy, a thyroidectomy, an excision of the rectum, these are not the occasions on which the anæsthetist can let the callow student handle the anæsthetic even under supervision. Consequently the student is reduced merely to watching, and in a practical matter like the administration of an anæsthetic watching is little better than no teaching at all. It is the actual management of the patient that is all important. The anæsthetist in hospital

has to do his best for the patient and for the surgeon. It is unfortunate that so often this is just what is of least use for the student so far as preparing him for his own subsequent practice is concerned. The ideal condition would, of course, be that in all surgical practice, private as well as hospital, the anæsthetic was handled only by an expert. At present that is far from being an attainable ideal. Many men in ordinary practice are of necessity obliged to give an occasional anæsthetic. It is tragic that there is so little opportunity in their undergraduate years for really preparing them for this ordeal. It is true that the number of anæsthetists employed in hospital, both visiting and resident, has increased and is increasing. Consequently there is an increase in the number of expert men available all round. This number is, however, not nearly large enough to cover any but a small part of the whole field, although it is large enough to make difficult the earning of a decent living in certain regions. Indeed the remuneration of the specialist is almost as difficult a problem at the present moment as is the adequate training of the student. A way will have to be found to improve both of these functions.

We know that the Association of Anæsthetists is giving full attention to the former subject, that already they have had some success, and that there is good prospect of still further improvement being obtained. The training of students is not a matter with which they can so easily interfere. It must be left to the teachers themselves to solve this problem, with the existence of which we know that they are fully aware.

## ANÆSTHETIC TWITCHES

By F CARTWRIGHT, M R C S , L R C P , D A \*

**M**ANY things conspire together to break the calm of the operating theatre, the patient may strain, he may vomit, he may try to climb off the table, while in the dental extraction room a morning of battle and murder may take the place of that silent and peaceful atmosphere which characterizes the textbook description of gas anæsthesia

Besides these gross manifestations of the imperfections of anæsthesia or its administration, there are minor troubles which interfere with the surgeon's manipulations. Some of these are common, some rare, some are important and some of no significance at all. The object of this article is to draw attention to a few of these curious phenomena.

First let us consider nitrous oxide jactitations. Actually these are a sign of asphyxia and the gas only causes them in that it deprives the patient of oxygen. The jactitation commences usually as a light tremor of the hands, arms, or legs, followed by twitchings of the facial muscles, spasm of the glottis and generalized clonic convulsions. Usually, but not always, the patient is deeply cyanosed and often the eyes are rotated outwards with the pupils fixed and dilated. If oxygen or air be now admitted, the jactitation will quickly cease and the patient be none the worse. Their importance lies solely in the fact that they are a sign that a dangerous degree of asphyxia is approaching, and it is in practice safe, in the straight gas used for extracting one or two teeth, to bring the patient to the level of commencing jactitation. It is understood that there are some practitioners so far gone in sin that they look upon jactitation as the cardinal sign of gas anæsthesia.

This form of twitch, though in not so marked a degree,

\* By courtesy of *King's College Hospital Gazette*



is seen in any form of anæsthesia where the oxygen requirement is not being fully met. The usual signs are tightening of the muscles, spasm of the glottis and a light tremor. Probably the wandering "athetoid" movements of a limb or hand seen in gas oxygen anæsthesia are of the same nature. The remedy is to look to the airway and supply more oxygen.

Next we have a series of twitches of which ether clonus or ether tremor is an example.

This tremor is seen typically in robust young men, usually the "tough," the athlete who has begun to run to seed and is now out of training, drinking a little too much and smoking too many cigarettes. Induction is difficult with a wild excitement stage, and the patient commences to quieten down, but does not become settled. He is still rigid, his breathing is stormy and irregular and there is usually some laryngeal spasm. Presently (usually when one has just heaved a sigh of relief and announced that the patient is ready to go into the theatre) a fine tremor of the limbs develops and may become so violent as to shake the whole trolley, but it remains a tremor pure and simple and does not develop into anything remotely resembling a convulsion. If an attempt is now made to flex the arm, it will be found to be very resistant, and the clonus will be greatly increased by efforts to bend the limb. This infuriating, but quite innocuous, phenomenon occurs only in light anæsthesia and is probably a prolongation of the excitement stage. The only treatment is to carry the patient to a deeper level of anæsthesia, for which purpose it is frequently necessary to substitute the more powerful chloroform for the relatively weak ether.

A similar tremor is sometimes seen during evipan anæsthesia and during recovery from avertin. There is, as yet, no satisfactory explanation of this form of twitch, and it is harmless, but annoying. One type of clonus, not of anæsthetic origin, must also be mentioned here. It is a coarse trembling of one limb seen especially in patients in the lithotomy position, and is caused by stretching of the muscles due to a faulty posture. The remedy is obvious.

Finally we approach the milk in the coconut, the most

intriguing of all problems, the very vexed question of ether convulsions

This rare but appallingly fatal condition is characterized by twitching of the facial muscles occurring during deep and prolonged ether anæsthesia, the twitching rapidly spreads to the limbs and trunk until all the muscles are involved. This generalized twitching quickly becomes so violent as to form a true clonic convulsion which may actually necessitate holding the patient down to the table.

At the same time the respiration is seriously embarrassed by spasm of the vocal chords and by spasm of the intercostal muscles, and it is probable that the diaphragm is also involved. The convulsions usually stop with dramatic suddenness and in somewhat less than half the reported cases there is an uneventful recovery. In the remaining cases (estimated at about 60 per cent) death occurs more or less suddenly from circulatory failure at times varying from a few minutes to a few hours after the cessation of the convulsions.

These convulsions occur in a very definite type of case. The subject is usually young, suffering from a disease causing sepsis and high temperature, and having marked acetonaemia. Cases are more common in hot weather, in overheated theatres, and with the patient in the Trendelenberg position. Several of these factors are almost invariably present.

The following nine cases are either taken from the Hospital anæsthetic death book, or are cases occurring in my own experience or reported to me.

#### *Case One*

J C Age 17 Operation for osteomyelitis of tibia 18/6/33

Temp 104 Anæsthetic Gas, oxygen and ether

Induction commenced at 3 p.m.

3 10 Trembling of limbs Ether discontinued and gas oxygen substituted Hypodermic injection of morphia gr  $\frac{1}{8}$  Trembling ceased

3 25 Trembling recommenced Injection repeated

3 35 Generalized convulsions Intratracheal oxygen and CO<sub>2</sub> was given The respiratory rate was seven per minute

3 45 An intravenous injection of morphia gr  $\frac{1}{8}$  and glucose was given

4 10 Respiratory rate, 13 Rectal temp 109

4 15 Sudden death

There is no post-mortem report The inquest verdict was "Death due to operative shock, the anaesthetic being in no way to blame"

#### *Case Two*

M Age 31 Operation for ruptured ectopic pregnancy 24/7/34  
Temp ? Anaesthetic Chloroform ether mixture to ether (Open)

The operation had been in progress about 30 minutes when the patient developed wild convulsions Ten c.c. of calcium gluconate (Sandoz) was injected intravenously The convulsions ceased dramatically about a minute later and the patient made an uneventful recovery

#### *Case Three*

P Age 20 Operation for acute appendix 20/11/34

Temp 101 Anaesthetic Chloroform ether mixture to ether (Open)

Mild twitching of the facial muscles was followed by tremor of the legs towards the end of the operation This quickly ceased with no other treatment than inhalation of CO<sub>2</sub>

A sample of blood was taken and the serum calcium found to be 10.6 mgm per 100 c.c. of blood

#### *Case Four*

E.S. Age 53 Panhysterectomy for infected carcinoma 26/12/35

Temp 102 Anaesthetic Gas, oxygen and ether

Induction commenced at 10.20 a.m.

11.20 Breathing became shallow and gasping Colour and pulse were good Twitching of the facial muscles developed Coramine and intratracheal oxygen and CO<sub>2</sub> was given

11.55 Patient died suddenly

This operation was performed in the Trendelenberg position The theatre temperature was over 80 throughout

At post-mortem the lungs were found to be engorged, and the brain to be engorged and oedematous

The ether was analysed and found to be pure

#### *Case Five*

D.J. Age 2, 11/12 Operation for acute double mastoid 6/3/36

Temp 101.6 Anaesthetic Ethyl chloride and gas oxygen ether

Induction commenced at 6.40 p.m.

7.15 Twitching of the legs and face developed The ether was turned off and the twitching disappeared

7.25 The ether was turned on again

7.40 Twitching of right hand developed

7 45 Marked convulsions of the whole body Coramine and intratracheal oxygen and CO<sub>2</sub> were given The pulse failed, respiration became sighing, and at

7 50 Death occurred

At post-mortem there was found collapse of both lungs, right sided cardiac dilatation and marked congestion of the brain

#### *Case Six*

J C Age 31 Perineal repair 18/3/36

Temp ? Anæsthetic Gas oxygen and ether

This case had had two previous uneventful anæsthetics

Induction commenced at 2 15 p m

3 45 Convulsions commenced A hypodermic injection of morphia gr  $\frac{1}{4}$  was given The ether was turned off and oxygen and CO<sub>2</sub> given

4 Morphia gr  $\frac{1}{4}$  was repeated and coramine given

4 15 Coramine repeated The convulsions became weaker and the patient became pulseless

4 35 Death occurred

At post-mortem there was found slight congestion and collapse of the bases of both lungs, congestion of the cerebral vessels, hæmorrhage into the lateral ventricles and slight right-sided cardiac dilatation

#### *Case Seven*

R S Age 5 Operation for osteomyelitis 28/4/37

Temp 104 Delirious Anæsthetic Ethyl chloride to ether (Open)

Fifteen minutes after the operation commenced, twitching of the legs developed, leading to generalized convulsions The ether was discontinued and gas oxygen substituted The convulsions ceased after about seven minutes and the child made an uneventful recovery

#### *Case Eight*

C W Middle aged Operation Hysterectomy 20/5/37

Temp 97 Anæsthetic Gas oxygen and ether

Half an hour after the operation commenced, the patient developed typical ether convulsions A hypodermic injection of morphia gr  $\frac{1}{4}$  was given CO<sub>2</sub> was administered, and the table raised from the Trendelenberg position The convulsions ceased ten minutes later and the patient made an uneventful recovery

On the evening of the operation the mouth temperature was found to be 100 It had fallen to normal by the next morning

*Case Nine*

D W Age 40 Panhysterectomy for fibroids .26/5/37

Temp 99.8 Anæsthetic Gas, oxygen and ether

In addition to the usual atropine this patient received a preliminary hypodermic of morphia gr  $\frac{1}{8}$

Induction commenced 2.10 p.m.

3.20 Convulsions commenced in the face and spread all over the body in the course of a few seconds Morphia gr  $\frac{1}{4}$  was administered hypodermically, oxygen and CO<sub>2</sub> was given and the table was raised from the Trendelenberg position

3.45 Intramuscular coramine 17 c.c.

3.50 The convulsions ceased

4 Intravenous calcium gluconate 5 c.c.

4.40 Signs of circulatory failure 17 c.c. of coramine was given, and two minims of adrenalin into the heart Artificial respiration was commenced

4.50 Death

5 The rectal temperature was found to be 104

At post-mortem there was found extreme congestion of the pial vessels, but no petechiæ or hæmorrhages into the brain The right heart was dilated There was extensive basal collapse of the lungs with a few petechiæ There was marked congestion of the liver, spleen, kidneys, nail beds and lips

One or two striking facts emerge when we consider these cases First we find that out of a total of nine cases occurring over a period of four years, two occurred in March 1936, and no less than three in the month April 28th to May 26th, 1937 Secondly, we find that no less than four of the cases occurred in one theatre (gynæcological)

Five of the cases had a temperature of 101 or over, and, of the remaining four, three were operated on in the Trendelenberg position, while the fourth had an anæsthetic lasting an hour and a half in the gynæcological theatre before the convulsions developed

Next we come to the matter of the temperatures after the convulsions had been in progress for some time Here we find that the first case had the extraordinary temperature of 109, and possibly more, since that was the limit of the clinical thermometer employed The eighth case was found, in the evening of the operation, to have a temperature of 100, which had fallen to normal the next morning,

while the ninth case was found, after death, to have a rectal temperature of 104

Finally, there is the rather interesting position of cases two and three. Both these cases occurred in my own experience, one in this hospital and one in another. The first, a woman with an ectopic pregnancy and therefore, presumably, with a lowered blood calcium, received an injection of calcium gluconate with (apparently) dramatic effect. From case three blood was taken immediately the convulsions had ceased and the serum calcium was estimated, when the figure was found to be normal.

Let us see how these facts fit in with the theories which have been propounded to explain the cause of this puzzling disaster. All these theories are pure conjecture, most of them based on casual observation of one or two cases. It must be remembered that these convulsions are rare and, when they do occur, the anæsthetist's anxiety is such that he has little stomach for even simple investigations.

### (1) *Impurities in the Anæsthetic Agents*

The fact that we have one group of two and one group of three cases in our list might lend support to this theory. Again, the fact that ether was used in every case is a point in favour. Against this theory is the fact that the ether in this hospital is bought in sufficient quantities for a month's supply, and all the other agents are also bought in bulk, so that one would expect rather more cases of convulsions. So far analysis has failed to detect any impurity in suspected samples of the following: ether, ethyl chloride, gas, oxygen and atropine. There is also, against this theory, a reported case (Hudson) of an infant who developed a condition indistinguishable from ether convulsions while being operated upon under local anæsthesia. A rather intriguing suggestion has been made that the impurity has not been suspected and so not considered in the analysis, and is, in fact, a new impurity arising from the modern mass production of ether which was started at about the same time that ether convulsions were first described in 1926.

(2) *Abnormalities of the Blood Chemistry*

Acidæmia has been suggested as a cause. The supporters of this theory suggest that the starvation and vomiting which accompany so many cases of high temperature causes a lowering of the bicarbonate content of the blood producing an acidæmia which is increased by breathing the  $\text{CO}_2$  laden air which is found under the mask even in so-called "open" ether.

In our series we can find five cases of high temperature to support this theory, but little else in its favour. It is, however, a fact that the lipid soluble anaesthetics tend to turn the normal patient temporarily into a diabetic.

Alkalæmia has, rather unfortunately, been also suggested as the cause. Over-breathing washes out the  $\text{CO}_2$  from the lungs and consequently from the blood. This over-breathing may result from the stimulation of the ether vapour during light anaesthesia or stimulation of the respiratory centre by a high external temperature. It has been pointed out (Hoseason) that intubation also tends to lower the  $\text{CO}_2$  content of the alveolar air, but this is an argument against this theory, as most reported cases of convulsions have not had intratracheal anaesthesia. Another point against this theory is that the convulsions appear typically during deep anaesthesia, when the breathing is depressed. For the theory is the fact that cases are most frequent in hot, moist theatres and that alkalæmia probably lowers the ionized calcium in the blood and so tends to produce tetany which in some ways resembles ether convulsions.

Calcium deficiency is the latest theory to be advanced. This supposes that the serum calcium, already depressed by sepsis, is still further lowered by the factors mentioned under alkalæmia. It is pointed out that it is the ionized fraction of the blood calcium which is lowered and that many patients who show symptoms of tetany have a normal blood calcium. This probably discounts any evidence which might have been expected from the blood calcium estimation performed in case three. In support of this theory, we have the dramatic result of an injection of cal-

cium gluconate in case two, but calcium was of no avail in case nine, and the only other reported case in which it has been used (King) also failed

### 3 *Idiosyncrasy*

Heat Stroke "Fibrillary twitchings of muscles and convulsions usually result," says Price's Textbook of Medicine when speaking of heat hyperpyrexia. In Ross you will find the post-mortem appearances of sunstroke described as follows "Extreme venous engorgement especially of the cerebrum. The left ventricle is contracted and the right dilated. The lungs are congested. The liver and kidneys show cloudy swelling." Cases four, five, six and nine show this appearance at post-mortem and nothing else. In support of this theory we have the pre-existing high temperature in cases one, three, four, five and seven, also the fact that cases four, six, eight and nine occurred in the gynæcological theatre, which is notoriously the hottest and most humid theatre in the hospital. In addition we have the fact that the Trendelenberg position was employed in cases two, four, eight and nine, a posture which would tend to increase the congestion of the brain, which is the cause of death in heat stroke.

Other factors in support of this theory are the use of mackintosh coverings and the preliminary hypodermic of atropine, both of which diminish heat loss. Further evidence is also provided in cases one, eight and nine, in which there was a marked rise in temperature during or previous to the convulsions.

Unfortunately, there are several points against this very pretty theory. Firstly, the whole process is too quick. It seems almost inconceivable that anybody could be killed by a body temperature of 105 (case nine, rectal temperature 104) in the space of an hour. It might be argued that the swift rise of temperature from normal to 105 might prove fatal, but that leaves us with the query, Why does the temperature rise so suddenly? Secondly, it may quite justifiably be argued that the convulsions themselves are sufficient to cause the rise in temperature, leaving us no further than we were before. Thirdly, it seems curious



that the surgeon, wearing far more clothing, covered with a mackintosh apron and doing far more muscular work, does not succumb more frequently than the patient

Atropine idiosyncrasy has been put forward to account for these objections, and large doses of atropine do cause convulsions, but one would expect some sign of atropine overdose before starting the anæsthetic

### *Treatment*

It is at once obvious that, with so many conflicting theories of causation, any attempt at rational treatment is out of the question. Dramatic "cures" have been claimed for a variety of different methods, cures which have, in other cases, failed to produce any effect whatsoever. It must be remembered in this connection that, in a quite large proportion of cases, spontaneous and sudden cessation of the convulsions occurs and the patient makes a complete recovery without any treatment at all.

The various methods of treatment may be divided into three classes —

(1) *General* No attempt is made, in this line of treatment, to stop the convulsion. The heart's action is supported by the injection of a stimulant such as coramine, the cyanosis is combated by inhalation of oxygen with the addition of  $\text{CO}_2$  to stimulate the depressed respiration, and an intratracheal catheter is usually passed to overcome the intermittent spasm of the vocal chords which causes obstruction of the airway. From the point of saving the life of the patient this is probably the most satisfactory form of treatment at the present time.

(2) *Treatment of the cause* It is obvious that any attempt to treat the cause must depend upon what one believes the cause to be.

Impurity of the ether would be combated by the administration of  $\text{CO}_2$  to cause over-breathing and so rid the body of as much ether as possible in the hope that the toxic agent will be eliminated therewith.  $\text{CO}_2$  would also be administered by believers in the theory of alkalæmia.

Acidæmia is combated by administration of glucose, of

insulin, or better of controlled doses of insulin and glucose. This treatment enjoyed great popularity a year or two ago (glucose was administered intravenously in case one), but has now gone out of fashion.

Hoseason advocates the use of calcium gluconate (10 c.c. of the 10 per cent solution), believing the cause to be calcium deficiency. A point in favour of this treatment is that calcium diminishes neuromuscular irritability and is therefore presumably "good for convulsions."

Heat stroke would be treated by cold spongeing, ice to the head, rectal injections of ice water and venesection. Good results have followed treatment of the congestion of the brain (which may or may not be due to heat stroke) by raising the head of the table (this may have been successful in case eight) and by momentary compression of the carotid arteries (this failed in case nine).

Atropine idiosyncrasy might be treated by the injection of pilocarpine, though I know of no case in which it has been tried.

(3) *Symptomatic treatment* "We know that the convulsions cause the temperature to rise, which causes increased congestion, which causes convulsions, which cause the temperature to rise. We must break the vicious circle somewhere and, as we do not know the initial cause of the convulsions, let us break it by stopping the convulsions." Such might be the very reasonable argument put forward by a protagonist of the symptomatic treatment. The intravenous injection of morphia, of nembutal and of evipan sodium have been suggested to stop the convulsions. The hypodermic injection of morphia used in cases one, six, eight and nine is probably of little use, since it takes nearly half an hour to act.

Evipan is the best drug to use, since its action is short, but a very grave criticism of this type of treatment, whatever drug be used, is that it acts by depressing the neuromuscular mechanism to such a degree that convulsions are impossible. It is very questionable whether there is ever justification for the administration of a depressant drug to a patient who is not only deeply anæsthetized but also in

danger of death from respiratory and cardiac failure. However, there is at least one reported case (Chadwick) of recovery following the exhibition of evipan. The great point in favour of this treatment is that the convulsions are definitely stopped, the surgeon can get on with the job and so, provided the patient can recover from the effects of the drug injected, his chances of ultimate survival are greatly increased.

So much, then, for ether convulsions. It is in some ways a stimulating subject, since there are so many possibilities, and in other ways depressing, for, whatever treatment we employ, it appears that the patient will die in spite of all our efforts in some cases and in others will recover without a sign that our treatment has been of the least assistance to him.

In most cases where I have quoted from a writer, I have mentioned his name in the text, but I have also consulted, for the general principles, the writings of Nosworthy, Langton Hewer, Samson Wright, and Burridge. I am indebted to those members of the anæsthetic staff of the hospital, both present and past, who have given me permission to use their cases.

## TOXIC JAUNDICE FOLLOWING ADMINISTRATION OF PENTOTHAL

By J M VAIZEY, M D , M R C P

*Medical First Assistant, London Hospital*

**T**OXIC jaundice has long been recognised as a sequel to chloroform anæsthesia,<sup>11</sup> but the occurrence of this complication following intravenous anæsthesia with barbiturates does not seem to have been recorded. This is no doubt partly due to the fact that these anæsthetics were only introduced comparatively recently, but even more to the fact that the complication is much less frequently seen than with chloroform.

Toxic jaundice has very occasionally<sup>1 12</sup> been seen after repeated doses of phenobarbital (luminal) by the mouth, but recent reviews<sup>6 8</sup> of a large number of cases anæsthetized with evipan and pentothal do not mention this complication, though they discuss other immediate dangers and disadvantages. The following example of toxic jaundice following pentothal appears worthy therefore of record.

*Case Report* E W (41332/1937 L H), aged 44 years, single. A military hat trimmer. She was first admitted to the hospital in 1928 complaining of dyspnœa, palpitations, and tingling pains in the calves and feet. At that time a moderately severe anæmia (haemoglobin 55 per cent) of the hypochromic type was discovered. She was treated with iron and improved.

In 1934 she was re-admitted for rectal bleeding, and an operation for hæmorrhoids and anal fissure was performed. This was carried out under nitrous oxide and ether anæsthesia after premedication with omnopon and scopolamine. The patient made an uninterrupted recovery though she was

still anæmic (hæmoglobin 62 per cent) on discharge from hospital

Although there was no further visible rectal bleeding, her anæmia persisted, and in 1936 she was again admitted with dyspnœa, palpitations, and severe anæmia (hæmoglobin 36 per cent) She again improved on iron, but her blood did not become completely normal, and after her discharge from hospital the anæmia again increased in spite of continuous treatment with iron

It was decided, therefore, that she must still be bleeding from her hæmorrhoids, and in August 1937 she was re-admitted for operation

*Physical condition* She was a slight, sallow woman with marked pallor of the skin and mucous membranes The tongue was furred but showed no obvious glossitis, and the throat was clear No abnormal physical signs were detected in the heart, lungs, abdomen or nervous system, but on proctoscopy two small hæmorrhoids were seen

On August 25th she was operated upon and the two hæmorrhoids were ligatured The patient received omnopon gr  $\frac{1}{2}$  and scopolamine gr  $\frac{1}{150}$  as premedication and 0.6 gr of pentothal in a 10 per cent solution during the operation Nothing unusual was noted at the time of the operation

The next day the patient complained of much nausea and retching, but did not vomit A cloud of albumin appeared in the urine for the first time, but otherwise there were no abnormal signs

On the following day slight jaundice of the conjunctivæ was noticed, and this rapidly increased during the next week, being maximal ten days after the operation The liver became palpable and tender at this time and the urine was bile-stained, but the stools were never pale She was treated on a high carbohydrate diet with 10 c.c. of calcium gluconate intramuscularly on four occasions, and also with calcium lactate by the mouth, and made a complete recovery

	On admission (16 Aug 1937)	On discharge (26 Sept 1937)
Blood-count (red blood-corpuscles)	3,300,000	4,400,000
Hæmoglobin	56 0%	73 0%
Colour index	85	88
Polymorphonuclear leucocytes	61 5%	63 0%
"          lymphocytes	26 0%	22 5%
Large hyaline cells	11 5%	17 0%
Eosinophil cells	0 5%	1 5%
Basophil cells	0 5%	—

The red cells were seen to be large and pale in all the stained films. The total white cells were not counted on either of these two occasions, but on September 8th they numbered 12,000.

Van den Bergh reaction. Immediate direct reaction, positive, bilirubin, 4.0 mg in 100 c.c. on August 30th, 1937.

Lævulose tolerance test. Fasting blood-sugar, 0.08 gm per cent, 45 gm lævulose then given, 45 minutes later blood-sugar, 0.118 gm per cent, two hours later blood-sugar, 0.08 gm per cent.

Radiograph of gall bladder showed no evidence of gall-stones.

Here, therefore, is a patient 44 years of age with anæmia and hæmorrhoids who developed toxic jaundice following anæsthesia with pentothal. Though it is possible that this was simply a coincident "catarrhal jaundice," her age, the time at which it appeared, and the coincident albuminuria, make it more likely that the anæsthetic was responsible for the jaundice.

*Discussion.* Pentothal sodium (sodium ethyl 1 methyl thiobarbiturate) has been used extensively in the United States<sup>a</sup> and more recently here<sup>b</sup> also, as an intravenous anæsthetic, and yet toxic jaundice has not apparently been recorded. Whether this particular compound which contains sulphur will prove more toxic to the liver than other barbiturates remains to be seen, but experimental work with other barbiturates has shown that their effect on the dog's

liver in therapeutic doses is negligible,<sup>3, 4</sup> and this remains true even if the liver has been previously damaged<sup>5</sup>

Pentothal sodium and other intravenous barbiturate anaesthetics are generally regarded as unsuitable in cases of liver damage,<sup>7</sup> presumably because of the more markedly depressant effect on blood-pressure which they produce in such cases owing to slow detoxication<sup>9</sup>

In the present instance there was, however, no clinical evidence to suggest liver damage before the operation took place, but there were two factors present which are generally considered to contribute to its production by anaesthetics. These were anoxaemia and pre-operative morphine, both of which<sup>2, 10</sup> increase the risk of damage to the liver after ether, nitrous oxide or ethylene anaesthesia

In the present case, anoxaemia was inevitable as the patient had anaemia, and morphine was given both by the mouth to ensure constipation and by injection as omnipon before the operation. How far these were important as contributory factors it is impossible to say, but certainly other equally anaemic patients have received similar premedication and then pentothal without becoming jaundiced, so they cannot be paramount

It is remarkable, too, that this patient showed none of the more usual symptoms of intoxication by barbiturates, such as mental confusion or cutaneous eruptions, and no haemolysis of red blood-corpuscles was seen

*Summary* A case of toxic jaundice following pentothal anaesthesia is described and the condition is briefly discussed

In conclusion, I wish to thank Dr R A Rowlands for permission to publish this case, Dr P N Panton for the laboratory reports, and Mr N A Gillespie for much valuable advice and criticism

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## SOME OBSERVATIONS ON PENTOTHAL SODIUM

By C P DIXON, M B , B S (Lond )

**I**N observing the effects of anæsthetics, the ideal conditions are those in which one has a standardised operation, one that would be sufficiently painful to the patient to gauge the depth of anæsthesia. In such an operation as in skin grafting a radical mastoid cavity one has these conditions. The patients are all moderately healthy adults, and the cutting of the Tiersch graft provides a standard stimulation which is a suitable gauge of the patient's insensibility.

For this purpose twenty-patients had evipan sodium given intravenously, with omnopon gr  $\frac{1}{2}$ , and scopolamine gr  $\frac{1}{150}$  as premedication, given 30 minutes previously. Eighteen patients had pentothal sodium, with the same premedication.

In observing the induction stage one saw that all patients having pentothal sodium seemed to show a more sudden change in respiration. In two cases there was a short period of apnoea with slight blueness, but both breathed satisfactorily without our resorting to artificial respiration. In one patient having evipan, an asthmatic, respiration ceased, the patient went blue, an intratracheal tube was passed and oxygen was given through the rest of the operation.

In the evipan cases there was some movement of the legs and contraction of the quadriceps muscle as the graft was being cut. In only one of the eighteen having pentothal sodium was there any quadriceps contraction. In both series of cases the patients were sufficiently deep for the insertion of the graft, the average duration of operation being about twenty minutes.

In observing the after-effects, the patients who had had evipan sometimes came round rather boisterous and un-

controllable The patients having pentothal tended to pass into a deep sleep and wake up after a few hours as if waking from natural sleep It is difficult to draw conclusions from this type of observation as the personal idiosyncrasy of the patient both to the anæsthetic and to the premedication must be taken into account, but the impression was gained that the post-operative effects of pentothal sodium are the better

Reviewing the relative merits, pentothal sodium is an anæsthetic which, from the observation of these cases, has no greater risk than evipan, and gives a greater degree of anæsthesia as shown by enabling a Tiersch graft to be cut without stimulating the quadriceps, and has possibly slightly better after-effects

One unpleasant but apparently not serious disadvantage is that it appears to be an irritation to the subcutaneous tissues, and if being given subcutaneously, there is a painful reaction in the subcutaneous tissues Two cases were seen in which the drug was given subcutaneously and the arms treated with fomentations afterwards

When seen about a week after the injection, both patients complained of tingling down the anterior surface of the forearm, and of a painful lump in the ante-cubital fossa On examination we found that there was a hard tender nodule, apparently fibrous, in the subcutaneous tissue at the site of the injection They complained of tingling of the arm between the elbow and the wrist, and were found to have very considerable loss of sensation over a narrow strip of skin about two inches wide from the elbow to the wrist joint, presumably due to involvement of the volar branch of the anterior cutaneous nerve of the forearm In one case, after three months the nodule was still palpable, but not painful, and there was no hypoæsthesia

## EVIPAN SODIUM IN DENTAL SURGERY.

By JOHN BUNYAN, L D S

THE writer has used this anæsthetic for various dental operations constantly for over four years. The following points are stressed on its use —

(1) It is inadvisable to use evipan in the surgery owing to the uncertain time needed for recovery.

(2) There is rarely need for any pre-anæsthetic, but one tablet of evipan given the night before and also one hour before helps with the very nervous patient. Three bromide tablets gr 15, are also of use.

(3) The arm should be immobilised in a simple straight splint. This precludes the possibility of the arm moving and causing the needle to jump out or through the vein. Such details as the size of the needle and the type of syringe are of importance.

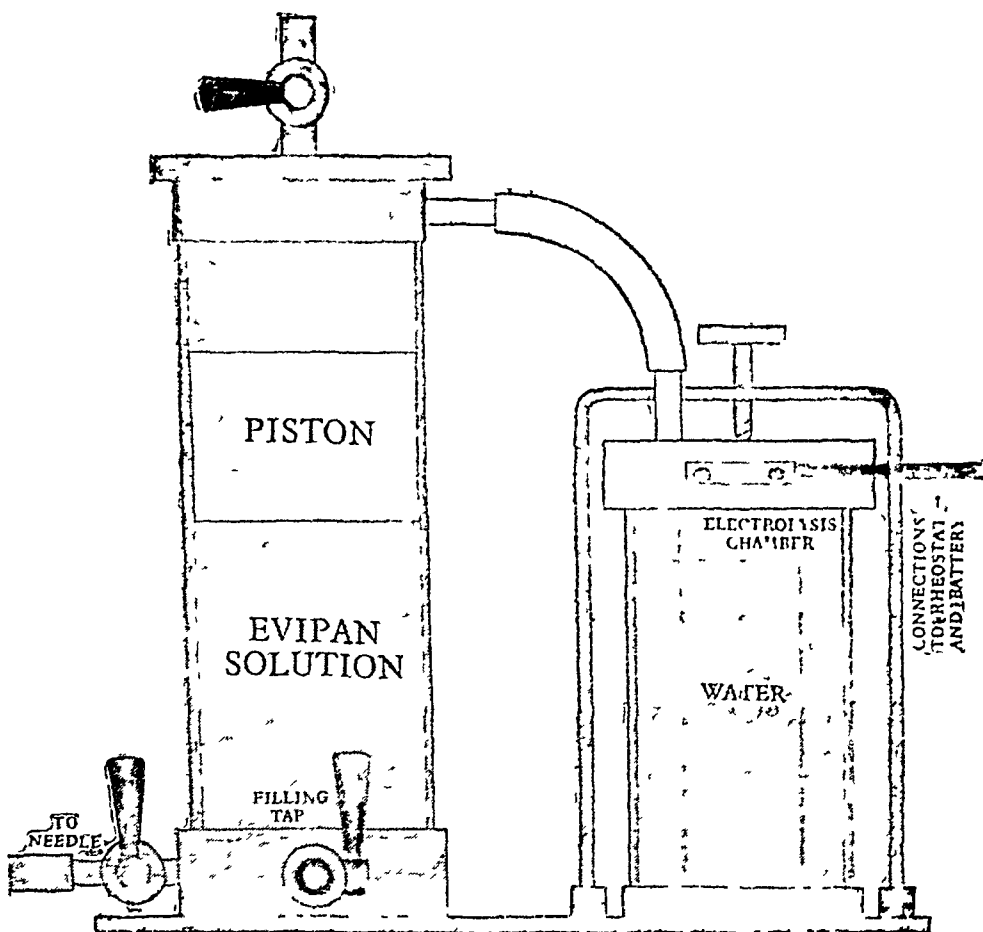
(4) A mouth prop must be inserted before the injection is started, as the masseter is rarely relaxed.

(5) The eyes should be covered as light acts as a stimulus and shortens the period of anæsthesia.

(6) Owing to the cough reflex being returned during evipan anæsthesia, it is not possible completely to pack off the throat, but a taped sponge held by the site of the operation and one at the back of the tongue are quite effective in preventing the swallowing of blood.

(7) After the operation the patient is immediately put back to bed on his side with the knees up, and allowed to sleep on without any disturbance.

(8) In over 2,000 cases there have been no cases needing a cardiac stimulant and in only one case has CO<sub>2</sub> been used.



The bulk of the writer's work is performed in the country, where the services of the skilled anaesthetist are not available. The only alternative to evipan is chloroform and ether, with all their attendant risks and disadvantages. Evipan brings the advantages of a first-class anaesthetic within the reach of the general and dental practitioner without the expense and inconvenience of complicated apparatus. The advantages claimed in using evipan are —

- (1) Its use obviates psychic trauma to the patient
- (2) The induction is pleasant and rapid
- (3) During the operation the patient is quiet. The surgeon is not hampered by the anaesthetist, and owing to the length of anaesthesia obtained, is able to take his time
- (4) The degree of haemostasis is better than with any other anaesthetic
- (5) Post-operative sickness is rare
- (6) In the writer's experience there has been no case of pulmonary complications
- (7) Recovery is complete within four hours

In order to make continuous injection of evipan a more certain procedure, the author has designed and used the following apparatus, known as the *evipanometer*. The principle is simple, in that pressure from the electrolysis of water in a small chamber is applied to the piston and can be varied immediately at will.

The apparatus may be mounted on the arm or on a table alongside the patient. It has given anaesthesia up to 2½ hours using 22 c.c. of evipan solution.

In the past there have been many arguments against the use of evipan. Firstly, that it was not controllable. Whilst it is true that once the solution is in the vein it cannot be taken away again, we have found no cause to necessitate its removal. After all, once chloroform or ether has caused respiratory, or particularly, cardiac embarrassment, the amount breathed in by the patient cannot be eliminated.

It has been suggested that evipan is responsible for cases of agranulocytosis, as far as we can tell none of the 2,000 cases has shown any signs of anæmia.

To sum up, in the author's opinion, evipan sodium is the ideal dental anæsthetic, owing to its simplicity, pleasantness and safety

## CHIN RETRACTION A NEW SIGN IN ANÆSTHESIA

By J U HUMAN, M R C S , L R C P , L D S

*Anæsthetist to St Mary's Hospital, Plaistow, and to the  
Metropolitan Ear, Nose and Throat Hospital*

THE value of a sign in anæsthesia depends upon its reliability, the ease with which it can be noticed, and the constancy of its occurrence

In chin retraction we have a sign which is the easiest of all the signs to observe and which, since I first discovered the level at which it occurs about 18 months ago, has appeared in over 50 per cent of the 600 cases which I have anæsthetised to this depth since then










Chin retraction has, of course, been noticed by most anæsthetists and I believe some call the phenomenon "tracheal tug," but no importance has been attached to it except to regard it as an accompaniment of good third-stage anæsthesia

To the best of my knowledge the level at which this sign occurs has never before been described I cannot find any reference to it in the literature on anæsthetics

The sign consists of a downward movement of the larynx and chin with each inspiration, but as the thyroid cartilage cannot be seen in many patients, I prefer to call it chin retraction, as this movement is more easily seen In cases where the chin does not actually move, the tightening of the chin depressor muscles can always be either seen or felt just below the chin by the anæsthetist's fingers Usually the chin moves visibly

In the accompanying diagram it will be seen that the third stage of anæsthesia is divided into four planes In the first plane the pupil is small, the corneal reflex is still present, and the eyeball is active At the bottom of the

first plane eyeball activity ceases, the corneal reflex disappears, and the pupil now begins to dilate. It is here that chin retraction begins, and as the anæsthetist always holds the mask with one or two fingers under the chin, he can feel the rhythmical tightening of the muscles under his fingers. It enables one to place the degree of anæsthesia

	MOVING EYEBALL	CHIN REFLEX	CORNEAL REFLEX	PUPIL
1 <sup>st</sup> PLANE				
2 <sup>nd</sup> PLANE				
3 <sup>rd</sup> PLANE				
4 <sup>th</sup> PLANE				

THIRD STAGE OF ANÆSTHESIA

here without looking at the patient at all. This sign remains active and obvious throughout the further deepening of anæsthesia, and in ascending anæsthesia it stops abruptly at the upper level of the second plane. The corneal reflex in ascending anæsthesia is unreliable and generally does not return until the upper border of the first plane is reached. The contraction of the pupil when anæsthesia rises back into the first plane is very constant, but it is always only a relative sign. The respiration tells one nothing when one is trying to distinguish between first and second plane anæsthesia.

It seems that the most reasonable explanation of the mechanism of the chin retraction would be that the muscles which by their tone hold the larynx in position, relax at the bottom of the first plane so that with each downward movement of the diaphragm the lungs are drawn down-



wards bodily for about a centimetre, carrying the trachea and larynx with their attachments downwards with them

It occurs whether ether, chloroform, or nitrous oxide is being given, although it is not often that second plane anæsthesia is reached with  $N_2O + O_2$  alone. I have, however, come across chin retraction in obstetric anæsthesia on three occasions, and once I have even seen it while giving nasal gas and air for tooth extraction

Chin retraction is always more marked when a closed system of anæsthesia with rebreathing is employed

I have found this sign particularly useful in operations about the head and neck, where the eyes are generally covered with sterile towels, and even where the chin and neck are covered over, the movement of the chin can usually be seen through the towels. If not, a hand advanced under the towels and placed upon the neck will detect rhythmical tightening of the chin depressor muscles if anæsthesia has descended below the level of the first plane

First plane anæsthesia is, as a rule, all that is required for operations about the head and neck, and the anæsthetist is often able to watch, from the other end of the operating room, that anæsthesia does not become unnecessarily deep by noticing chin retraction when it first occurs

## THE DIPLOMA IN ANÆSTHETICS

The diploma was granted in October to Mr J F Mackenzie, R. M. Muir, and T T Stobbs.

At the November examination held by the Royal College of Physicians and Surgeons the following candidates were successful and have been granted the Diploma (D.A.):

- A H L Baker, LMSSA, Oxford and St. Mary's Hospital, W County
- Eva G Byrde, MB, BCh (Camb), L.R.C.P., MRCS, St. Mary's Hospital, W County
- J C Buckley, L.R.C.P., MRCS, Camb. and Westminster Hospital, W County
- Bessie E Cook, MB, ChB (Man), L.R.C.P., MRCS, St. Mary's Hospital, W County
- Ellen B Cowan, MB, ChB (Glas), Glasgow General Hospital, Glasgow
- M H A Davison, MD, BS (Dur), Royal Victoria Hospital, Newcastle
- A J S De Freitas, L.R.C.P., MRCS, St. Bartholomew's Hospital, E Suffolk and Ipswich
- L M De Silva, L.R.C.P. & S (Edin), L.R.C.P., MRCS, Colombo General Hospital, Colombo
- S F Durrans, L.R.C.P., MRCS, Cambridge and St. Mary's Hospital, East Dorset
- Florence Faulkner, MB, ChB (Man), L.R.C.P., MRCS, St. Mary's Hospital, Manchester Northern Hospital and Manchester Royal Infirmary, Manchester
- W B Gough, MB, ChB (Birm), L.R.C.P., MRCS, St. Bartholomew's Hospital, G Gray, L.R.C.P., MRCS, St. Bartholomew's Hospital, G Gray, L.R.C.P., MRCS, St. Bartholomew's Hospital, G Gray, L.R.C.P., MRCS, St. Bartholomew's Hospital
- J R G Harns, MB, BCh (Camb), L.R.C.P., MRCS, Cambridge and St. Mary's Hospital, East Dorset
- J K Hasler, MB, BS (Lond), L.R.C.P., MRCS, St. Mary's Hospital, East Dorset
- Ursula Y Im Thurn, L.R.C.P., MRCS, University College, London
- Freda C Kelly, L.R.C.P., MRCS, University College, London
- J O Mofiat, MB, ChB (Glas), Glasgow Royal Infirmary and St. Mary's Hospital, East Dorset
- D A Prothero, L.R.C.P., MRCS, St. Bartholomew's Hospital, G R Rawlings, MB, BCh (Camb), L.R.C.P., MRCS, St. Thomas's Hospital

W H Scriven (Captain R A M C), L R C P, M R C S, Cambridge and King's College

E W O Skinner, L R C P, M R C S, St Mary's Hospital

G C Steel, L R C P, M R C S, Kent and Canterbury and Middlesex

O Walker, M B, Ch B (Liver), St George's Hospital and R A M College

We think it may interest many of our readers to see the questions which were set for the written part of the examination on this occasion. They were as follows —

(1) Write an account of the methods by which ether has been given in the past, and state how it is used at present

(2) Give a detailed description, with a diagram, of the opening of the larynx as seen through the laryngoscope when used for endotracheal work. What structures may be damaged when the laryngoscope is used, and how can such trauma be guarded against?

(3) Describe the action of cyclopropane, and state how it is usually administered

(4) You are asked to anaesthetize a baby for an operation to relieve pyloric stenosis. State what anaesthetics may be used and describe how they should be given. Indicate your choice

(5) How would you give a local anaesthetic for a tracheotomy? What changes may be observed in the respiration after a tracheotomy? Give reasons for such changes

## ASSOCIATION OF ANÆSTHETISTS GREAT BRITAIN AND IRELAND

THE Annual General Meeting was held on November 30th, 1912, at the Medical Society of London, Chancery Lane. Officers were elected for the ensuing year: President, Dr. C. H. H. Blomfield, Vice-president, Dr. C. H. H. Blomfield, Secretary, Dr. H. W. Mennell, Members of Council, in place of the late Professor R. R. Macintosh, Dr. I. W. M. Nosworthy, and Dr. V. E. Vassell.

After grateful reference had been made to the members of Council, the President also mentioned the deaths of Dr. H. P. Fairlie and Dr. H. G. G. G.

The Council's report for the year showed that it was being given to the conditions of work for anæsthetists when employed in connection with operations in the paying wards of hospitals to which the anæsthetist is attached. A sub-committee to deal with this matter had been set up and had reported to the Council. In connection with this a statement had been drawn up which was available on request for any anæsthetist. Further consideration had taken place on the whole question of anæsthesia, remuneration in private practice, and in consequence of resolutions passed at the general meeting representative of the Association were to confer with the Consultant Group of the British Medical Association. The Council noticed that without instigation from the Association a well informed article had appeared in the general Press stating views much in accordance with those of the Association. The new regulations of the Central Midwives Board for instruction in obstetric anæsthesia had been reported to the Council. During the year 12 new members had been

elected, and Lord Nuffield and Dr R M Waters, of Wisconsin, U S A , had been elected honorary members

The Treasurer's report was then read and showed the finances of the Association to be in a thoroughly satisfactory state. The Treasurer was thanked for his care and for the great help which he provided to the secretarial work of the Association.

We understand that since the general meeting representatives of the Association have been invited to and have attended a session of the Consultants Group of the British Medical Association. After a statement of their views by the delegation a long discussion took place, and a resolution was eventually passed by the members of the Group present. This resolution was fully in accordance with the desires of the Association's delegates, who expressed their gratitude. It is hoped and believed that future action based on the resolution will be equally satisfying.

## CORRESPONDENCE

*To the Editor of the British Journal of Anæsthesia*

## SECONDARY SATURATION

Westminster Hospital,

October 20th, 1937

Dear Sir,—Dr Harris's article in the July number of the *British Journal of Anæsthesia* is unfortunate in that the account of Secondary Saturation selected for discussion is that of a critic, and is one that does not in any sense represent the aim and object of its supporters

According to McKesson,<sup>1,2</sup> the brain cell derives its oxygen from a liquid where the dissolved nitrogen amounts, roughly, to 17 vols per cent and the oxygen to 0.24 vols per cent. An average of 18.84 vols per cent of nitrous oxide was found to produce anæsthesia in man<sup>3,4</sup> and about 25 vols per cent in dogs,<sup>5,6</sup> where it is more constantly accompanied by cyanosis. The ratio of 7 molecules of nitrogen to 1 of oxygen is compatible with normal physiology, but when 100 molecules of nitrous oxide are present to 1 of oxygen, anæsthesia results. This may be due to the dilution or dispersion of the oxygen molecules, so that certain cells are unable to find enough oxygen with which to maintain metabolism or chemical activity up to the usual rate.<sup>1,2</sup>

Without accepting the view of gas anæsthesia given above, it is clear that the presence of any nitrogen in the alveolar air will diminish the amount of nitrous oxide that can be given in a mixture containing enough oxygen. Nitrogen will be present in some degree in alveolar air until it has been entirely displaced from the body. The view of McKesson and others<sup>1,2,5,7</sup> is that nitrous oxide, being more soluble than oxygen in the tissue fluids, will the more rapidly displace nitrogen. This is probably a fallacy. The

only factor determining the rate of elimination of nitrogen, apart from the blood flow, is the gradient from the tissues to the alveolar air. Provided there is no rebreathing of expired nitrogen, it will be eliminated equally rapidly whatever inert gas is administered, whether nitrous oxide, oxygen, hydrogen, or any other. The only way to accelerate elimination in practice is to increase the pulmonary ventilation. According to McLeod,<sup>5</sup> complete desaturation will take about two hours, though the first half will be removed in about 25 minutes, mainly from the more vascular tissues.

In practice it is found that nitrous oxide anaesthesia deepens a little after some time, but any more rapid deepening produced by the McKesson technique must be due to some other factor, which may well be as in the passage quoted by Dr Harris.

Saturation with nitrous oxide, too, will take place not a great deal more rapidly from a 100 per cent than from an 80 per cent mixture, and complete equilibrium will not be reached within the average duration of a nitrous oxide anaesthesia.

It is unlikely that anyone would deliberately injure the patient by suboxygenation in order thereby to increase his susceptibility to anaesthetics. A low percentage of oxygen is used only to allow of a high percentage of nitrous oxide. Those who permit a certain degree of cyanosis may be wrong, but they do so believing that no injury results. While clinical observation seems to point to a higher vomiting rate in patients who have been cyanosed, in a great many cases, especially after short administrations, where slight cyanosis has been allowed in the presence of a free airway, there is no detectable injury.

Yours, etc.,

GEOFFREY ORGANE  
(M B, D A)

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### ANALGESIA IN

To the Editor

Dear Sir,—I read with much concern, the letter written by you on the above subject in the October *Journal of Anaesthesia*. I, in my certain points which appear not to answer the criticisms

(1) The figures that were given of labour pains refer to estimations by palpation, and the patient's sensation has been made, and a good number of 60 seconds and over, both in the first and labour. These pains were timed by palpation, and the period of uneasiness which follows was not included

(2) This period of uneasiness must be covered by method of relief in order to satisfy the requirement of patient. There is no irrelevance about this fact

(3) The irregular, prolonged, or rapidly succeeding pains of the late second stage constitute one of the conditions for which the gas and air analgesic method is primarily provided. In the hands of midwives nothing is allowed by the Central Midwives Board, and, in my opinion, under such circumstances, no stronger anaesthetic agent is necessary or advisable

(4) I have not found, in my experience, that the symptoms induced by the inhalation of two or three deep breaths of pure gas continue for some 70 or 80 seconds



Since reading Professor Chassar Moir's letter I have obtained one of his apparatus, and carried out investigation with it, which can be described under two headings

(a) The effect of experiments conducted on myself, and also on Dr R Penn Harbord, Demonstrator in Anæsthesia, University of Liverpool His report, which agrees with what I found, follows "Two or three breaths of pure gas produced the first sign of analgesia in 16 seconds This was five to eight seconds quicker than the standard gas and air machine In 30 seconds the analgesic effect was the most intense, it then declined gradually for 15 seconds and became practically absent in 60 seconds At 30 seconds the gas and air machine produced a state of analgesia as intense as the pure gas machine, but from this time onwards the intensity increased "

(b) The effect upon patients in the Liverpool Maternity Hospital This was observed both by myself, by Dr Harbord, and by the Labour Ward Sister We all agree that (1) There was a variable amount of relief when the pains were not long in duration nor severe in character, and this relief was more quickly obtained than with the gas and air apparatus (2) In many cases the analgesic effect of the gas wore off before the pain was over, and the patient complained towards the end of the pain in consequence This did not occur with the gas and air apparatus (3) In a number of cases there was some cyanosis after inhalation, which soon disappeared (4) There was no relief whatever from the inhalation of two or three deep breaths of pure gas in some cases

It is of interest to record that Dr Hilda Garry has had an opportunity of using the Chassar Moir apparatus personally, and makes the following statement "Two or three breaths of pure gas inhaled from the machine produced the symptoms of analgesia more rapidly than the gas and air apparatus, but the effect was not sustained long enough to relieve a pain of any intensity The relief obtained from gas and air, both in the first and second stage, was far greater "

Finally, upon the evidence at my disposal, I must dis-

sent from the statement that the results given by the pure gas method of analgesia is any improvement on that obtained with the nitrous oxide and air mixture. Attention must be drawn, however, to the right technique to be employed for the latter, and I cannot do better than quote the following from a paper, published in the *British Medical Journal* on September 11th, 1937: "In order that the full effect be appreciated it is important that the mask be applied to the face on the slightest indication of a pain, viz., before it has become established. The patient should be instructed to inhale and exhale normally, and to increase the depth and frequency of the breathing in proportion to what she can feel."

A patient who realises that a pain is going to be a severe one can inhale a gas and air mixture just as quickly and as deeply as she can fill her lungs with two or three deep breaths of pure gas. At most the effect obtained with the former is only delayed by five to eight seconds, after which the intensity of the relief is as good as the latter, and is increasing. It can then be enjoyed as long as it is required, and is applicable to the stage of labour for which Professor Chassar Moir—by his own argument—denies the use of his apparatus.

I remain, sir,

Yours faithfully,

R J MINNITT

---

#### UNIFORMITY IN APPARATUS

3A Ravenscourt Square,  
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To the Editor

Dear Sir,—For some years past we have become accustomed to an almost universal arrangement of rubber tubing on our anæsthetic tables and machines. Black tubing has been used for nitrous oxide, red for oxygen, and green for carbon dioxide. In the last year or so cyclopropane has been added to our battery of gases, and, more recently, white rubber tubing has appeared. These two newcomers

have been wedded and, at first sight, the partnership seems to be suitable

However, I have recently observed white rubber tubing applied to oxygen cylinders and, on questioning the arrangement, was told that as the gas manufacturers use a white label or a splash of white paint to distinguish oxygen cylinders, it is reasonable to use white tubing for the oxygen supply. The argument is presumably by analogy from the green paint and tubing of carbon dioxide cylinders. Now there has been more than one fatality in the past from the unwitting use of carbon dioxide instead of oxygen, and this confusion about cyclopropane conduction appears to offer further sources of error. It is obvious that no one should deliver to a patient a gas of any kind until he is quite certain what it is. Yet in hospital practice mistakes of this kind have been made by the relatively inexperienced and presumably will continue to be made. Therefore, sir, would it not be advisable for some authoritative body to make a ruling on this subject and to communicate its views to hospital authorities with a request that they should fall into line.

I am, sir,

Yours truly,

G EDWARDS

## ABSTRACTS

"*Advances in anæsthesia*" E B TUDHY in *New Orleans Med and Surg Journ*, September 1937, pp 146—7

THE author mentions two gases not often alluded to in articles on anæsthetics Trichlorethylene, trethylene, is, he says, a volatile non-inflammable and non-explosive agent Comparatively small amounts produce anæsthesia and there is danger, if induction is hurried, of respiratory embarrassment Relaxation is not so pronounced as that of ether, but recovery is more rapid Used from a sponge or handkerchief in doses of 20 to 30 drops trethylene is useful for allaying the pain of tic douloureux and of coronary disease Helium is the other gas alluded to This is said to be helpful in maintaining anæsthesia in cases of hyperthyroidism in which there is retrotracheal enlargement of the thyroid gland, or pressure on the trachea which restricts the size of its lumen

"*Toxic effects of carbon dioxide*" R M WATERS in *New Orleans Med and Surg Journ*, October 3rd, p 220

THE author, after giving an illustrative case in which death was attributed to carbon dioxide, discusses the effects of the gas on normal subjects Ten per cent appears to be the maximum strength that can be inhaled for more than ten minutes without loss of consciousness Above 12 per cent there is sudden decrease in respiratory stimulation Much individual variation is found in both subjective and objective effects, alarming symptoms appearing in one individual from a concentration tolerated by others Excess carbon dioxide inhaled by normal individuals causes hyperpnoea, progressive increase in systolic blood-pressure, and other alarming signs and symptoms until concentrations above 10 per cent are reached when unconsciousness uniformly occurs Depression of both respiration and circulation

eventually supervenes as the tension is increased. Inhalations of variable concentrations have often been followed by convulsive manifestations. In all experiments there has been extreme variability in the reactions found in different normal individuals, both animals and men. This raises the question as to whether diseased states may not have a definite influence on the sensitivity to toxic effects of carbon dioxide. There is indeed evidence that diseased states may modify still further individual variability. Persons toxic from acute infections and from dietary deficiency are often hypersensitive. Extremely hot weather, starvation, and dehydration seem to lead to greater susceptibility. The physical signs to be recognised as showing excess of  $\text{CO}_2$ , are either muscular activity, generally starting with twitches of the face, or signs simulating collapse, low blood-pressure, rapid pulse, pallor, and quick, gasping breathing. Treatment consists in maintaining a free airway and giving pure oxygen.

*"Anaesthetics from a hospital superintendent's point of view"* A. J. HUGO in *South African Med. Journ.*, August 28th, 1937, p. 563

THE author contributes to a symposium on anaesthetics. His recommendations are worth quoting: (1) That the anaesthetist be a highly trained man in his subject. (2) That he be a capable physician. (3) That he be one who gives full co-operation to and works in consultation with surgeon, physician, pathologist, hospital superintendent and nursing staff. He must work in sympathy with fellow practitioners whose patients come under his care. (4) That no patient be submitted to operation until the anaesthetist has carefully studied the patient's physical condition in consultation with surgeon, physician, pathologist and chemist. (5) That our universities and teaching hospitals give more care to the teaching of anaesthetics, so that men who qualify will be fully competent to give an anaesthetic safely, and so that hospital superintendents will not find that every time a new house surgeon is appointed the resident medical officer's service has to be re-organised in order that the new house-man may stand by and learn. (6) That general practitioners receive

facilities for post-graduate anæsthetic courses (7) That the anæsthetist avail himself of post-graduate special anæsthetic teaching and also keep in close touch with recent advances in medicine

*"Continuous nitrous oxide-oxygen-air analgesia in obstetrics"* R C SWORD in *Anæsthes and Analgesic*, October 1937, p 249+

THE title of this article is a little misleading, for the author's routine treatment of the patient at confinement is to give three to nine grains of nembutal after labour has started and within the next hour or so 100 to 150 gr scopolamine. The nitrous oxide administrations are not started till the first stage is over. They are carried out by the use of a mask which is illustrated in the article and which has a chimney-piece with perforations that can in certain positions permit air entry. The mask is strapped to the face and administration begins with the admission of 1,000 c c nitrous oxide and 200 c c oxygen. The author claims to have kept up analgesia for about 41 minutes to one and a half hours on an average, and that in about three-quarters of his patients actual expulsion of the foetus was happily performed without any descent into true anæsthesia. The anæsthetic was discontinued before the placenta was expelled. It is claimed for the method that it allows the mother to take the fullest advantage of every contraction after complete cervical dilatation and that no objectionable results have followed, either to mother or to child.

*"Local anæsthesia with supplementary intravenous narcosis"* P FELDWEIG in *Schmerz, Narkose Anæsthesia*, October 1937, p 107

THE author maintains that the greatest safeguard against thrombosis embolism and pulmonary troubles after laparotomy is the avoidance of general anæsthesia by inhalation. He gives statistics of 661 laparotomies in support of his view. His method is premedication half an hour before operation, then, a quarter of an hour before, careful infiltration of the line of the incision throughout its depth with

novocain suprarenin solution, and finally just before or after the peritoneum is opened an intravenous injection of evipan. In operations on women he generally finds that 2 c c are enough. The rapid awakening by the time the abdomen is closed is a great advantage over the conditions left by a long inhalation.

*"Rectal 'twilight-sleep' in labour"* P HAUPSTEIN and E GUNTHER in *Schmerz Narkose Anæsthesia*, vol 1, No 4, p 124

THE authors having abandoned Gauss's original method with morphia and scopolamine, but being convinced of the desirability of securing a similar result, have tried to obtain these by various rectal injections. Their article deals chiefly with rectidon, which is a barbiturate similar to pernocton, and with evipan. The accounts do not lead us to suppose that either of these agents given rectally supplies a valuable means of combating the pains of labour. The rectidon which gave some amnesia was often accompanied by violent excitement, and evipan, although certainly obliterating all pain for a time, produced no amnesia and required repetition.

*"Pentothal sodium as hypnotic in obstetrics"* MACPHAIL, GRAY and BOURNE in *Canadian Med Assoc Journ*, November 1937, p 471

THE article provides good ground for regarding pentothal sodium as a really valuable hypnotic in obstetrics. The authors compare its effects with those of other barbiturates, especially nembutal, and show that pentothal is free from the objections which can be found to the use of many of these drugs. There is no excitement associated with pentothal taking and the infant is unaffected. Moreover, the simple route, by the mouth, is perfectly effective. The duration of labour is shortened by pentothal, and the authors believe, though they cannot explain why, the cervix dilates rapidly and that it is in the first stage of labour that time is gained. They gave 4 gr of pentothal by the mouth as soon as pains were definitely established. Half an hour later another 3 gr

were given and this amount repeated after an hour. Usually the patient was by now quiet, sleeping between pains, and willingly doing the obstetrician's bidding. Two or three grain were given again every half to one hour if the effect appeared to be wearing off. The total quantity of pentothal used varied from 10 to 20 grains. With a view to enhancing amnesia 1/100 to 1/500 gr scopolamine was given at the time of the first dose of pentothal.

*"Changes in conditional responses, due to anaesthetics"*

S. DROVRKIN, WESLEY BOURNE and B. H. RAGINSKY  
in *Canadian Med Assoc Journ*, 37, 136-139, 1937

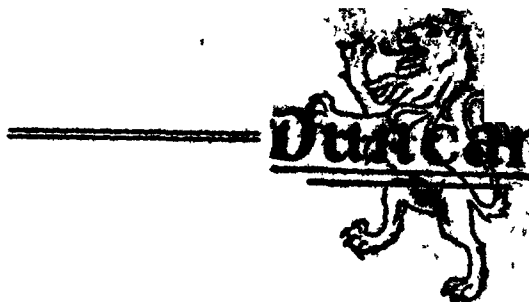
THE authors have based their work on Pavlov's observation that conditioned salivary reflexes could be modified by various drugs. They have experimented with dogs and cats. Summarising their results it may be said that alcohol, amytal, nembutal, avertin and paraldehyde were effective in weakening the processes of inhibition. Nitrous oxide and ethylene had a uniform depressing action. Morphine and hyoscine abolished positive responses, seemingly by producing nausea and loss of interest in the food.



## OBITUARY

WE regret to record the death, at the early age of thirty-two, of IVOR NICHOLAS LEWIS, M R C S , L R C P , Anaesthetist to St George's Hospital. Mr Lewis held anæsthetic appointments also at the Metropolitan and Brompton Hospitals, besides being busily engaged in the private practice of his specialty. He was a man with a lively outlook on his work and always ready to try new methods or drugs, and he contributed to our pages an article on avertin technique in thyroid surgery in the days when avertin was comparatively a novelty. Later, Lewis had specially devoted himself to endotracheal methods in the use of which he displayed the highest skill. Many readers will recall his contribution on the subject, only last September, to the *British Medical Journal*, an article which gave rise to considerable subsequent correspondence. It is deplorable that a career so full already of performance as well as promise should be ended at this tragically early stage.





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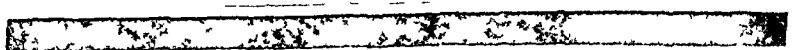
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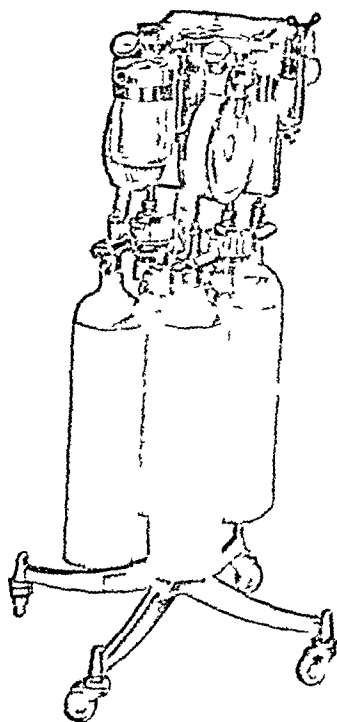


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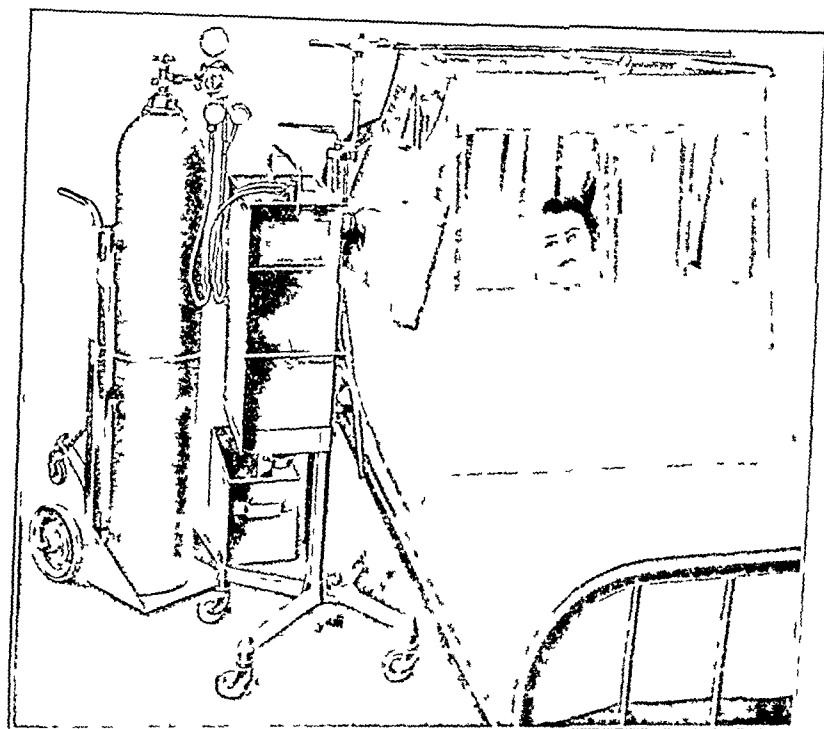
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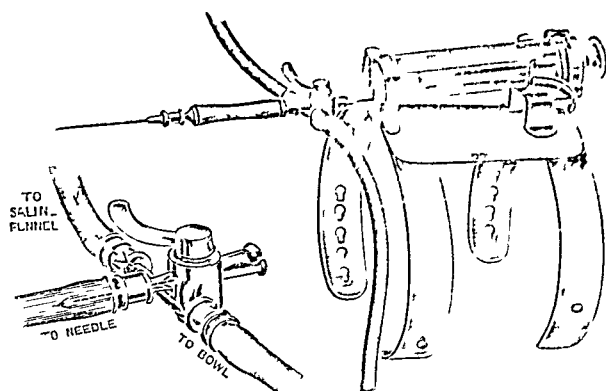
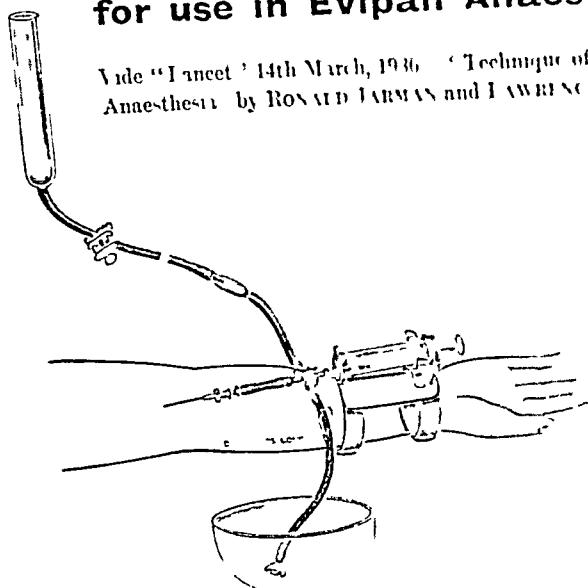
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Vide *British Medical Journal*, March 15, 1930, pp 458-9 and 495-6, and April 5, 1930, pp 669-70. *The Lancet*, March 15, 1930, pp 57, 4 and 557. *Proceedings of the Royal Society of Anaesthetists*, April and July, 1930, and January 1931. *Proceedings of the Royal Society of Medicine*, May, 1930, pp 919-25. *British Journal of Urology*, June, 1930, pp 129, 130, and 179. *Edinburgh Medical Journal*, April, 1931. *Strasbourg Medical Hospital Journal*, February, 1931, pp 9, 6. Etc., etc.

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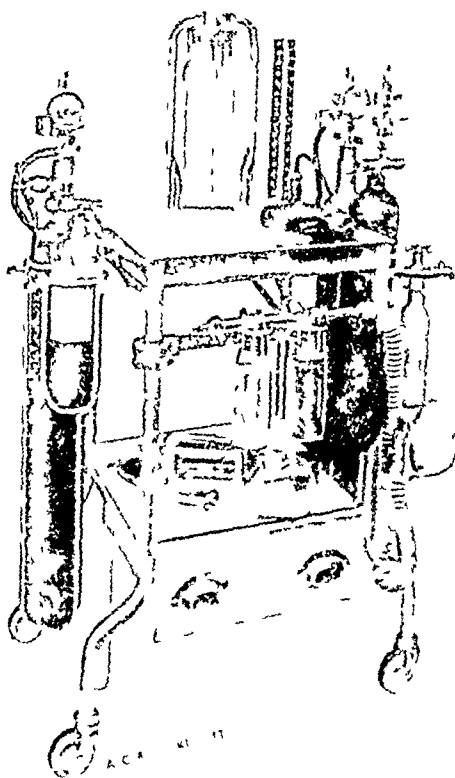
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# British Journal of Anæsthesia

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VOL XV No 3

APRIL, 1938

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## EDITORIAL

### THE ULTIMATE RESULT

WE have before now put forward the opinion that if fault is to be found in the development of anæsthetic practice as it is seen to-day that fault lies in our failure notably to reduce the fatalities of anæsthesia. We might produce statistics and figures to prove that this fault-finding is justified by the facts, and that the mortality rate among persons subjected to anæsthetics has not fallen in recent years or indeed that it has risen. We have, however, little faith in statistics when they are applied to the incidence of fatality or of any other single phenomenon in connexion with the administration of anæsthetics. Our opinion is based rather on observation of current writings on anæsthesia, on personal observation, and on the information supplied by colleagues. Assuming then that it is true that the mortality rate in their practice is not being lowered by anæsthetists, we would instigate them to make every effort to alter this state of affairs for the better. The great advances which have been made in the practice of our speciality are very obvious to anyone who watches expert anæsthetists at work. The condition of the patient during operation, no matter how severe or prolonged the operation may be, is something that could hardly have been imagined a generation or two ago. The quiet breathing, the utter relaxation, the well

sustained circulation, and the complete removal of any interference with the surgeon's manipulations from the proximity of the anæsthetist or his apparatus complete a picture which may well be called the surgeon's dream. The disturbing question is, though, are we painting this picture only at the price of a very different one on the reverse of the canvas? In other words, does the patient's condition afterwards often upset our satisfaction with what we saw at operation? In the discussion on massive collapse of the lungs which took place recently at the Royal Society of Medicine, Professor Grey Turner made some remarks very pertinent to the subject we are discussing. He observed that with the older methods of anæsthetic giving the patient coughed more and was probably more uncomfortable than he is to-day, but he was safer. Now, it should not be beyond the wit and skill of anæsthetists to secure safety without abandoning the modern progress. It may be contended that the fatalities nowadays are almost entirely after and not during operation, and that it is impossible to attribute them solely to the anæsthetic. Yet the number of deaths on the table is not negligible and it is these, when the subject is ordinarily fit and the operation not a grave one, which may fairly be reckoned against the anæsthetist. The cure for these is education and more education, for those fatalities do not occur with the expert.

## "DEATH ON THE TABLE"

An Address to Students by  
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THIS phrase is short and clear. The meaning appears to be obvious. It implies that a patient has died during the course of a surgical operation. Now, among the duties of His Majesty's Coroners is that of inquiring into the causation of such a fatality and death on the table usually results in the holding of an inquest. The occurrence has to be notified to the Coroner's officer. This official makes a few preliminary inquiries and reports the matter to the Coroner. The Coroner may or may not decide to order a post-mortem, he may or may not decide to hold an inquest and he may or may not decide to call a jury to assist him. In actual fact an inquest usually is held without a jury, and, for convenience sake, the post-mortem is made just before the assembling of the court. All this forms a simple, straightforward routine, which up to a few years ago was not so well-worn as it is now. At one time, if the poor wretched patient could be got off the table still alive, there seemed to be no need to report his ensuing death. One breath, only one, or the merest flicker of the pulse when outside the theatre door and no more was said. The phrase "death on the table" was taken absolutely literally. I well remember the first operating fatality I saw. The patient was a boy of about twelve who was being operated on for a cerebellar tumour. The occiput had been removed and the surgeon was probing the cerebellar tissue when the anæsthetist gave a warning cry. The skin flaps were hastily put back into position, a few stitches were put in to hold their edges together and a dressing was thrown on the wound. The House Surgeon then picked up the child and carried him out of the theatre trailing festoons of towels. The child—or, by this time one should say the body—was dumped on a trolley

and rushed back to the ward, which was about twenty-five yards away. There pulse and respiration were found to be imperceptible and it was announced that the child had died *en route* from the theatre. In fact, although the operation had not been a great success, the child had not died—on the table.

It is obvious that if deaths during operations and deaths under anæsthesia were to be investigated at all, such arbitrary arrangements about what were and were not cases for investigation were ludicrous. About twelve years ago Coroners began to require that all cases in which the patient failed to recover consciousness after an anæsthetic should be reported to them. That rule is now universally observed and so, unless we are to limit these discussions very severely, we ought to alter our title to "Death under Anæsthesia", which will include those cases which die actually in the theatre, those which fail to come round and those which die following the administration of spinal or local anæsthetics.

All of us here are, or are about to be, technically qualified to administer anæsthetics. If we live long enough we shall, at some time or another, be faced with the necessity of such an administration, and further, if these administrations be numerous enough, we will surely end by meeting with a "death during or from the administration of an anæsthetic". For most of you, the possibility is very remote. Some of you may have to give anæsthetics frequently, and will, in consequence, find yourselves wondering on occasion what on earth you will say to the Coroner if the object of your administration doesn't recover. He usually does recover. The unlucky few of you may become full-time anæsthetists and although you will not go about in mortal terror of "death on the table", there will be a number of near shaves—if not of actual disasters—alarming enough to give you plenty to think about whenever you are unfortunate enough to pass a sleepless night.

Now for figures. We are told that anything can be proved by statistics. It may be so, but as far as I have been able to investigate anæsthetic figures, it seems that nothing can be proved. The Registrar-General's returns for England and Wales have, since 1923, specified the number of cases in

which an anæsthetic was directly concerned in a death. You will see that in the twelve years 1923 to 1934 the numbers nearly doubled. On the face of it, this is a severe criticism of the present generation of anæsthetists. But we may observe that the figures were stationary for 1923-4-5 and then shot up phenomenally in 1926. That was the exact point at which the Coroners began to be more exacting in their views as to what should be investigated. This heavy increase was followed by further large advances in the next three years; also due, I think, to the yet increasing number of cases notified rather than to an increase in the actual death-rate. In the years 1930-34 the rate of increase slowed down. The numerical changes can easily be appraised when the figures are plotted as a graph.

Unless we have accurate figures, not only of the number of deaths that take place but also the number of anæsthetics administered, we cannot possibly judge as to whether anæsthesia is getting safer or more dangerous. A census of the number of anæsthetics given each year over a number of years is obviously impossible of attainment and we must investigate as far as we can on a smaller scale.

In this hospital during the five years 1923-27, 31 "deaths on the table" occurred, and all of them were "literal" deaths on the table—that is to say, the patients died in the theatre proper. For strict purposes of comparison we must reduce this figure to 30 as one of the fatalities occurred in the out-patient theatre and I have not compiled the number of anæsthetics given there. In the five years 1933 to 1937, 27 deaths took place, but six of these we must deduct as the patients died some time after their return to the wards—this time varying from 1 to 54 hours. We have, therefore, a total of only 21 cases to contrast with one of 30. The operations conducted in the theatres were 11,871 in 1923-27 and 13,821 in 1933-37. That is to say, operations went up by 16.5% whilst deaths went down by 30%. In other words, the chance of death in the theatre was reduced from 25 in 10,000 to 15 in 10,000—a reduction of 40%.

This sounds very pleasing. But things are not so simple as all that. In the first place, does the hospital have the same number of acute and desperate cases nowadays that it used

to have, and secondly, what changes, if any, have taken place in the hospital routine? To get some idea of the incidence of serious risks in the surgical practice of the hospital I have found the figures for the years in question of operations for strangulated herniæ. These show that the number of such difficult cases fell from an average of 29 per annum in the first quinquennium to 10 per annum in the second. So although our fatality numbers are down, we really have not had the dirty work to do that there used to be in the past. These cases, I imagine, are now largely admitted to the hospitals of the London County Council.

Secondly, the routine of the hospital has been altered. During the first of the periods we are contrasting, emergency anæsthetics fell to the lot of the Cottage residents—however experienced or otherwise they might be. Nowadays, if a case is to be operated on for any dangerous condition, the Honorary Anæsthetists expect to be, and are, rung up for duty and one of them attends. There is nothing more that I can give you in the way of statistics. Attempts to evaluate the relative safety of the various anæsthetic agents do not lead to more than the vaguest results. Even in the days when ether and chloroform were almost the only drugs used it was difficult to obtain a real contrast. Some cases, such as bronchial and nasal ones, could not have ether and were at the same time somewhat poor risks; these weighed down the evidence against chloroform. At the same time many of the ether cases had a late mortality from chest complications and these passed uncounted. In our modern routine, where even the simplest operation on a hospital list may call for an anæsthetic which starts with premedication and is completed by nitrous oxide, oxygen-and-ether, with possibly some chloroform and some carbon-dioxide thrown in, the task of evaluating the various factors is hopeless. If we could use nothing but chloroform for a whole year and then nothing but ether for another year, some reasonable information might be gathered; but such an arbitrary course is frankly impossible. Practical anæsthesia used to be looked upon as an art, it is rapidly becoming a practical science; but it will never achieve the clear-cut lucidity of an exact science. At most we can merely record that certain types of cases

do best with certain types of anæsthetic and bend our energies to producing the minimum amount of physiological disturbance

Let us look at the causes of death under anæsthesia. They are legion, ranging from death by frank overdose of chloroform to the death from acute cardiac failure which happens to take place just as a local anæsthetic is being given with a view to draining a pyopericardium. One large group, however, stands out—that due to simple errors—those cases, in fact, in which death would not have occurred had ordinary sensible precautions been observed. During the last ten years a number of these have been recorded in various parts of the country—they are surprising in their simplicity and avoidability. First, liquid anæsthetics have been squirted directly into the patient's pharynx, leading to immediate death when chloroform was used and to fatal bronchopneumonia with ether. The error here has been in reversing the leads to and from a Junker or Shipway apparatus. The only answer is that all such bottles must be tested before use. The more recent models of these machines are fitted with valves so as to prevent the possibility of such an accident. Even with this addition it is necessary for the anæsthetist using a Shipway to see that the ether and chloroform are in appropriate containers. Ether in the chloroform bottle will do no harm and very little good, but chloroform in the ether bottle may give a fatally high concentration of vapour. The latter, in any case, must not be so completely filled as to allow liquid to be splashed into the exit tube.

This possibility of the wrong filling leads to another type of case in which the wrong agent is unwittingly used. We cannot handle chloroform or its mixtures with the fine abandon with which we pour on ether, and it is necessary for us to be absolutely certain what liquid is contained in any bottle we are using. Not long ago in a provincial hospital, an anæsthetist asked a young theatre nurse to fill his ether drop-bottle. He took the bottle back from her and proceeded with his administration. The patient collapsed and was resuscitated. The anæsthetic was continued and once more the patient collapsed—this time finally. It turned out that either by misunderstanding or by carelessness the nurse



had filled up the bottle with chloroform. There was a lot of discussion about the case, especially when the poor girl was suspended from her work by the matron. In fact, the public protest was so vigorous that she was almost immediately restored to duty. This case warns us always to fill our own bottles or at least to watch whilst they are being filled.

There are cases in which ether or other inflammable gases are exploded and the patient's air passages are severely burnt. Whenever diathermy, cauterization or radiography is to take place, and whenever there is a naked flame or an open fire, ether, ethyl-chloride, ethylene, acetylene, and cyclo-propane are not to be used. This is well known and no one is likely to fall into this error. Not long ago, however, in one of the hospitals of East London, a man was being treated by cautery for an epithelioma of the mouth. Quite rightly he was anæsthetized with gas, oxygen and chloroform. During the operation his pulse began to fail and the operation had to be stopped. Amongst the restorative measures applied was that of insufflating him with ether vapour and oxygen, and by this or other means he was revived. The operation was then re-started. An immediate explosion took place in the patient's air passages and death occurred shortly afterwards from septic broncho-pneumonia. At this point it is well to remind ourselves that electrical connections and flexible wiring are always potential sources of sparks, either by improper adjustment or by wearing out of the coverings. All electrical appliances such as headlights and spotlights need to be kept in adequate repair. They are used very frequently and the risk of sparking is very small, so that we can hardly banish ether from the theatre on this account, but nevertheless we must not run into any danger from dilapidation of the insulating materials.

Failure of the oxygen supply during the giving of a gas and oxygen anæsthetic seems an easily preventable mistake yet it has occurred. They tell the story at one North London hospital of a resident anæsthetist who went for a smoke towards the end of a long and tedious operation. By the time he had found his cigarettes and had searched the changing room for a box of matches the patient was *extremis* from anoxæmia. The oxygen cylinder had run out.

at that particular moment. A more recent similar disaster took place in the North West of London. This time there was more excuse as the anæsthetist had left the theatre for the anæsthetic room to prepare the next case. No one giving a closed anæsthetic should leave his patient more than momentarily unless he is absolutely certain that his oxygen cylinder is full enough to run for some considerable time--unless, that is to say, the pressure-gauge, which should always be used, gives a reasonably high reading.

Last among these avoidable accidents, comes a new type of disaster. In a recent fatality in South London, an endotracheal tube had been passed, or was supposed to have been passed, preparatory to a tonsillectomy. It turned out that the tube was in the œsophagus. As the surgeon was about to begin the enucleation the patient was observed to be of a bad colour. The young anæsthetist hurried to turn on a fresh cylinder of oxygen and in the semi-darkness of the theatre opened it widely. The blast of oxygen which reached the œsophagus was of sufficient force to tear a hole in the stomach. This resulted in general peritonitis and death a few days later.

I have given you a melancholy recital and I have only dealt with cases in which a little more care applied to simple matters would have avoided disaster. There are two other sources of trouble which can be avoided by simple questioning of the patient. They are the dislodging of artificial dentures and the inhalation of regurgitated foodstuffs. We must always take care to examine the patient's mouth and to remove all plates and bridges. In hospital routine this is done for us in the wards with almost unfailing regularity the danger is more acute in nursing homes and in private houses. In hospital, we assume that cases for set operations have had no food for some hours before our anæsthetic started. Even so, we sometimes get remarkable ejaculations of half-digested fruit. There are some patients who, being deprived of their regular meal, proceed quietly to stoke up with the contents of their lockers or with what they can borrow from their neighbours. Should anything untoward occur in these circumstances then no one can be blamed but the wretched patient himself. Anæsthetics for emergencies

or accidents are a trap one may be so anxious to reduce a Colles fracture immediately one sees it that the fact of the patient's just having had a heavy meal is overlooked. This happened in the case of a Dulwich schoolboy who called at the tuck shop on his way to play football. In the first five minutes of the game he was injured and was rushed to hospital where gas was given at once to reduce his fracture. He vomited, inhaled his vomit and died. We must always inquire about meals in emergency cases and no general anæsthetic should be given within four hours of the taking of food—certainly never within three hours.

So much for the easily and obviously avoidable fatalities. There are two other easily avoided sources of danger which unfortunately are not obvious. Unless one has been told of them and happens to remember them, one can walk all innocently into appalling disaster. The first is concerned with the administration of nitrous oxide. Any swelling and, particularly, any inflammatory swelling of the tissues of the neck is an absolute contra-indication to the giving of nitrous oxide. I would mention to you goitre, grossly enlarged tonsils, quinsy, retro-pharyngeal abscess, inflamed glands and cellulitis of the neck, as cases in point. Marked congestion of the pharyngeal walls is normal in nitrous oxide anæsthesia and, in the presence of these conditions, it may produce a complete closure of air passages. The only hope, and that a forlorn one, is immediate tracheotomy. I remember reading of a young boy with quinsy taken to the casualty department of a provincial hospital. The resident on duty gave nitrous oxide with a view to opening the abscess, but the boy was dead after a few breaths. I also once saw the post-mortem on a man who was to have been operated on for an epithelioma of the floor of the mouth. He had massive secondary deposits in the cervical glands. The anæsthetist chose to induce with a Clover's gas-ether apparatus. There was no time even to start giving the ether. The other hidden snag lies in the fact that it is dangerous to use adrenalin when the patient is having or has already had chloroform. It may be perfectly all right, but cases of sudden and fatal collapse have been recorded and the risk must not be run. The peculiar thing about this matter is that no harm seems

to result if the adrenalin has been given first and then the chloroform second. Scores of nasal cases are reported where an adrenalin pack is put into the nose before the operation and where chloroform or its mixtures are used for the anaesthesia. No one has ever found that this method produced any increased danger, but chloroform, followed by adrenalin second is very risky.

Having sidestepped all these dangers, we are left with the possibility of death caused by toxic effect of the anæsthetic agents, or the effects of the operation, or a combination of both. Before our operation starts, however, we always have the possibility of death during the induction of anaesthesia—particularly during chloroform induction. There are the particular dangers of vagal inhibition, ventricular fibrillation in addition to the general risk of overdose. His Majesty's Coroners are well aware of the dangers of chloroform and we must never engage in it without some very good reason for not using one of the other agents.

Once the operation has started all kinds of things may happen. We may push our anæsthetic to dangerous depths, we may continue deep anaesthesia for too long a period, the operation may result in excessive loss of blood, sensitive tissue may be pulled on too heavily, muscle tissue may be badly lacerated, intestines may be too freely removed from the abdominal cavity, or the operation may encroach on the heart or the vital centres of the brain. The relative part played by such happenings, by the toxicity of our anæsthetic, and by the patient's individual condition cannot be assessed with any accuracy whatever, except in rare cases. If a healthy patient proceeds at once to die of chloroform poisoning—it must be the anæsthetic, if some other patient dies when the surgeon puts his knife into an aneurysmal sac, then it is the operation. Such clear cut issues do not often occur.

In all departments of medicine and surgery prevention is better than cure. This is particularly true of anæsthetic difficulties, for prevention of them is relatively easy, but cure is extremely difficult. Salvation lies in dealing with trouble before it arises and the only way to do this is to keep

a constant watch on the patient's pulse, respiration and colour—half of one eye being at the same time kept on the manœuvres of the surgeon. At the least sign of increase or irregularity in the respirations, or of cyanosis, immediate decision must be made as to what is wrong and steps taken to put matters right.

If the pulse fails, respiration will soon be impaired and the normal colour will fade to a ghastly grey. If breathing stops, the pulse will soon stop also and the patient will have gone dusky. If there is obstruction to respiration resulting in cyanosis, the respiratory movements will cease whilst the pulse will rapidly fall. Death is impending and frantic efforts must now be made and heroic measures must be applied. They may be successful, but the great thing is never to reach the point when they are needed. When a man tells a wonderful story of how by the most strenuous efforts he just avoided a death on the table, it is more reasonable to deplore the fact that the situation ever arose than to admire the dexterity with which he wriggled out of it.

Ten years ago when I first started to be an anæsthetist, the treatment of impending death in the theatre was rather different from what it is now. Gas-and-oxygen machines were hardly in use at all, endotracheal anæsthesia had only just been heard of, there were no reducing valves, carbon dioxide supplies were not available and coramine had not been put on the market. If trouble arose the first intimation was that the anæsthetist called for "oxygen". The moment the surgeon heard this ominous cry, he stopped his operation and started artificial respiration by squeezing on the patient's chest. This he kept up until the patient recovered or until he himself was exhausted, when the house surgeon took over. An oxygen cylinder of the type you see standing in the corner of the medical wards was dragged in by a porter. Usually the key was missing, having been dropped in transit. When this had been retrieved one of two things happened: either the cylinder was found to be empty, or the force of the outflowing oxygen was so great that the cork was blown out of the attached bottle and a pint or so of dirty water shot into the air, falling in a generous cascade on the sterile and the dirty alike. It usually took two or three

minutes to get running a suitable stream of oxygen. The surgeon continued his artificial respiration, the anæsthetist meanwhile having lowered the head of the patient and seized the tongue in a pair of brutal crushing forceps. A theatre nurse would now try to get ready an injection of camphor. This camphor was in solution in olive oil and the poor patient would be found some minutes later tugging away for dear life at the plunger of a syringe, trying to draw up the thick solution through a fine hypodermic needle. If the patient in spite of these vigorous efforts still refused to breathe, some bystander was asked to slip a hand under the blanket and to dilate the anal sphincter. This provides a powerful respiratory stimulus and was sometimes effective.

By now the patient was either better or dead: if the latter was the case, an upper abdominal incision was made and cardiac massage was carried out through the diaphragm. In the few times I remember seeing this done it was quite ineffective. Other manœuvres I heard recommended are brisk rubbing of the patient's lips and the giving of a few minims of brandy. I was once bold enough to try the taste of the theatre brandy. O Death, *there* is thy sting! Intracardiac injections of camphor and strychnine were also given, but the pathologists observed that they rarely reached the myocardium.

Nowadays, our first concern, if either respiratory or cardiac failure shows itself, is to pass a wide-bore endotracheal catheter. This is a quickly performed manœuvre, provided the catheter and spatula are ready at hand, as they always should be. We must tip the table so that the patient is in the Trendelenburg position: this corrects the cerebral anæmia consequent upon falling blood-pressure. Artificial respiration is now carried out in the assurance of effectiveness. It may be done externally by Sylvester's method, but is probably more effective when applied internally by forcing a mixture of oxygen and carbon dioxide through the endotracheal catheter. If external squeezing of the chest be carried out it must not be too vigorous. At a certain dental hospital one morning an old gentleman collapsed under nitrous oxide anæsthesia. He was submitted to artificial respiration and was resuscitated sufficiently to be

moved and admitted to the wards of a general hospital near by. Later he died, and at the post-mortem it became evident that the efforts of his would-be savers had resulted in the cracking of sixteen of his ribs.

If the internal method is used a mixture of carbon dioxide and oxygen is supplied from the gas-and-oxygen machine and this is forced through the endotracheal catheter by a temporary raising of the pressure, easily produced by closing the expiratory valve and squeezing on the re-breathing bag. This should be repeated at intervals of a few seconds. The proportion of carbon dioxide should not exceed 10 per cent, too vigorous a stimulation of a failing respiratory centre is most injudicious. I am reminded of a houseman who was called upon to give an anæsthetic for a diathermy. He wisely chose chloroform. Half-way through the operation the patient began to breathe very inadequately and to go blue. The chloroform was stopped and a blast of pure carbon dioxide was delivered into the mouth. Respiration at once recovered and, lest the patient should come round too far, more chloroform was given. Again the patient went blue and breathing stopped and again pure carbon dioxide was given—but this time without response. You will probably agree in thinking that the giving of oxygen with both the chloroform and the carbon dioxide might have led to a happier outcome. I must remind you here that certain gas-and-oxygen machines, of the type which give an intermittent flow, have by-pass taps to the oxygen cylinders. By suitable adjustment of the machine, it can be arranged to give so strong a blast of oxygen that when the face-piece is applied the patient's chest is forcibly inflated. This is often a most effective proceeding but such machines are not used to any great extent for general surgery in this country.

So much for our direct aid to respiration. Without it our attempts to maintain the circulation will be useless, and without a restoration of the heart's action our efforts to encourage breathing will be wasted. Each, of course, helps the other, but direct efforts to stimulate the heart's contraction are essential. Since the introduction of coramine practically all other drugs have been abandoned, with the

exception of pituitrin and icoral. Coramine is pyridine-beta carbonic acid di-ethylamide and can be injected hypodermically, intramuscularly, intravenously, and *in extremis* intracardiacally. The usual dose is from one to five cubic centimetres of the standard solution provided. It was introduced first as a respiratory stimulant, which it is, but as it is now understood to have a direct stimulating effect on the myocardium it is used for this purpose primarily. If death is impending five c.c.'s intravenously or one c.c. intracardiacally must be given—the hypodermic and intramuscular routes are useless if there is a deficient circulation. The practical point about injection into the left ventricle is that a needle at least four inches long is required. The collapsed heart falls well back from the chest wall and will not be reached by any ordinary hypodermic needle except in small children. The fine needles such as are now used for lumbar puncture are excellent for cardiac injection. Movement of the needle shows that the heart has been reached and that contractions are taking place. Cardiac injections have been used for many years and all sorts of drugs have been tried. The results seemed to have no relation to particular drugs, and the view has been put forward that it is the mere physical stimulus of the injection that has resulted in the re-establishment of the heart-beat. In other words, sterile water or even nothing at all would have been as effective. But whilst we are making the puncture we may as well use coramine, whose stimulatory effect may be of great value to both heart and respiration. When ventricular puncture of the heart has met with success, it has been observed that there takes place a series of ventricular systoles which do or do not give way to normal cardiac rhythm. If the auriculo-ventricular sequence does re-establish itself, we may recover our patient. If it does not, the ventricles will go into fibrillation and all will be lost.

A new cardiac puncture needle has been devised by which the auricle can be reached. It is, as you see, a curved needle and is passed through the third right intercostal space close to the sternum and is directed so that its point should come to lie behind the sternum and about four inches deep to it. Here the auricle should be encountered, and the prick of



the needle is expected to stimulate auricular contractions, and to re-start the heart-beat. Even if auricular fibrillation should arise, some sort of circulation may be established. I have not had occasion to use this needle in the theatre, but we have been using it for injecting dyes into bodies in the post-mortem room to observe the exact place to which the point may be expected to go.

If cardiac injection fails, then the heart must be massaged at once. An upper abdominal incision is made: the surgeon's hand is passed between the liver and ribs and the heart is squeezed up against the chest wall, which is held by the other hand. If access be easy, the diaphragm and the pericardium may be opened and the heart actually grasped in the massaging hand. In children the mere squeezing of the heart against the ribs can be carried out through the belly wall without making an incision.

*Time* is the great factor. The longer the heart has been dead the less likely it is to be resuscitated. That is of importance, but even more vital is the question of the cerebral circulation. The cerebral cortex is extremely easily killed, and if circulation ceases for more than three minutes it is almost certain that the cortical cells will never recover. Time and again we read of cases in which cardiac injections and cardiac massage have restored the heart-beat, have re-established the circulation and have re-started regular breathing, but the patient has failed to recover consciousness and has died anything from a few minutes to two and a half days later. Those wretched "living corpses" lie there with a poor circulation, with gasping respiration, with half-open eyes, dribbling mouths, and, to the eye of those who have seen such things before, no earthly hope of recovery. Of course, they are witnesses of our heroic efforts to save them and we are praised in nice little paragraphs in the evening newspapers, which tell us how wonderful it is that we can restore the apparently dead to corporal existence even though it is unconscious and merely temporary.

So, gentlemen, we have seen how to avoid unnecessary accidents and we have discussed briefly the current method of attempted resuscitation. There are two other matters we have to consider. The first is "status lymphaticus," and the

second "ether convulsions" The first has had more wild discussion than anything else, I should imagine, in the whole field of medicine and surgery, and more widely divergent causes have been suggested for the latter than for any other clinical phenomenon on record

"Status lymphaticus" has had its very existence denied by a joint commission of the Medical Research Council and of the Pathological Society of Great Britain and Ireland. At the same time status lymphaticus has been cited as leading to anæsthetic fatalities when the variation from normal of the lymphatic tissues has been almost unobservable It has been "brought in," as it were, on no more evidence than was the old inquest verdict of "temporary insanity" Because the man took his own life, then he must have been temporarily insane, and because the patient died suddenly under anæsthesia it must have been a case of status lymphaticus

If the body at post-mortem shows (a) a markedly large thymus, (b) hyperplasia of lymph tissue throughout, especially in Peyer's patches, and (c) fatty degeneration of the heart-muscle, there is enough abnormality to allow the anæsthetist to excuse himself with the plea of the lymphoid state

Where these conditions have been found after an anæsthetic death it nearly always appears that the death had been entirely unexplicable and unexpected and that nothing was done by the administrator that was not usually quite safe In other words, the patient behaved in an entirely unpredictable manner and no one could be blamed In the actual occurrence the patient is usually a child, and the anæsthetist is faced with a sudden primary cardiac failure, most often but not always during the early stage of induction If you should meet with a sudden, unforeseen collapse, and if your efforts to resuscitate the heart-beat meet with no success, then you may buoy yourself up with the hope that the pathologist will find a lymphatic hyperplasia On three occasions, patients have died in my hands without warning in one case I honestly expected that a hypertrophy of lymph tissue would be found, and it was in another case it came as a heartfelt and entirely unlooked for relief, and in the third I neither expected it nor did the post-mortem

reveal it To an anæsthetist "status lymphaticus" or "the thymic state" is a hidden terror in the living and a complete acquittal in the dead

"Ether convulsions" are very real things They are quite distinct from the tremor of light anæsthesia or from the clonus which is sometimes observed They appear when anæsthesia has been established for some time and start by a twitching of the face muscles which spreads to the body and to the limbs The abdominal twitching is so violent that it is quite impossible to close a belly wall and there may even be difficulty in keeping the patient on the table The muscular inco-ordination produced is sufficient to interfere with respiration, as the glottis, the intercostals, and the diaphragm all become involved These twitchings stop spontaneously sooner or later, but in half the cases death occurs from cardiac failure shortly afterwards All sorts of theories have been put forward as to the cause of these convulsions They include heat stroke from high fever or an unduly hot theatre, impurities in the ether, under-oxygenation, over-oxygenation, heating the ether, tetany from calcium deficiency, acidæmia, alkalæmia, cerebral congestion from prolonged keeping in the Trendelenburg position, cerebral anæmia from the anti-Trendelenburg position, in fact, nearly anything and nearly everything

We had a case in this hospital last week I was anæsthetizing a healthy young man for the adjustment of an old fracture of the radius He was put well under with gas, oxygen and ether Towards the end of the operation I began to reduce the concentration of the ether, but at the surgeon's plea that his teeth, I should not be allowed to come round before the plaster was set, I returned to full strength Just as the dressing was being applied tetanic spasms appeared in the arm, and then in the leg which was hidden under the operation towels was found to be twitching The abdominal muscles began to dance and there was no hope of keeping the bone in position, and I determined to let everything go until some other man got the patient got over the convulsions I stopped the ether at once and gave carbon dioxide and oxygen I was undecided whether to try to control the convulsions with chloroform or with Evipan

Mr Landaw, whom I called in from the adjacent theatre suggested the latter and prepared to give the Evipan which I had meanwhile ordered to be got ready. Just as he was about to inject the solution, the twitchings stopped. A few seconds later and we should have hailed a new success for Evipan. The patient then recovered uneventfully. I hope to describe the case more fully elsewhere. The main point is that, so far as I can see, there was no factor concerned other than the depth of the anæsthesia. If you should meet this distressing condition, stop your ether at once, give oxygen and carbon dioxide, and, if necessary, control the twitchings by the minimal injection of Evipan-sodium intravenously.

Gentlemen, whenever you are so unfortunate as to have a death on the table, you will find that your mental reactions are of great interest. You may be convinced that you are the greatest criminal unhung that you have been guilty through negligence of nothing less than absolute bloody murder. On the other hand, you may feel that you are the victim of a fatal conspiracy, that the patient whom you have been trying to revive was actuated by a wilful perversity and was determined to die on you, that nothing you or anyone else could have done could have prevented it. The real truth will be somewhere far from either of these extremes. It may be elicited at the post-mortem and the inquest, but not necessarily. Some things are too involved to be clear, and you, the pathologist, and the coroner may all take different views of the same facts—none of you right and none of you wrong.

What I recommend you to do is to make written notes, immediately after the death, of the exact times of starting the anæsthesia, of the patient's collapse and of the end of your efforts at resuscitation, and to check these times with the surgeon and house surgeon. Though the facts may be irrelevant and unimportant, it does not do for the various witnesses to differ in their recollection of what exactly happened.

Finally, I hope that what I have said may be useful to you if ever you meet with death on the table, but even more strongly I hope that you never will have that misfortune.

## NEMBUTAL-CHLORAL NARCOSIS IN CHILDBIRTH

By F B MALLINSON, M R C S , L R C P , D A

*Hon Anæsthetist to the Woolwich War Memorial Hospital,  
and to St John's Hospital, Mavelen Hill*

SINCE her late Majesty Queen Victoria made her historic decision to have chloroform administered during the birth of her children, the view has been slowly but steadily gaining ground that the sufferings of parturition should be abolished or at least mitigated to the greatest extent compatible with safety to mother and child

When one considers the amount that has been and is being done to relieve pain and suffering generally in surgery (and also in medicine) it seems only rational to apply the same principles to the woman in labour

The argument often put forward that a mother cannot bear as great a love for her child does she not suffer the agony of its birth is surely too specious to bear investigation and savours more of mediæval superstition than up-to-date science

Agreement on the relief of pain in labour, however, seems now to be fairly general amongst our profession, as is shown by the number of drugs and methods tried out in recent times

The line of attack on this problem may be divided into two categories. What may be called the anæsthetic-analgesic and the amnesic-analgesic methods. Gas and air is the prototype of the former, while certain members of the barbiturate group of drugs illustrate the latter

The three main criteria in the technique of obstetric narcosis are.

- (1) Full amnesia rather than anæsthesia
- (2) The retention of a sufficient degree of voluntary control by the patient as to allow her to carry out simple instructions given her by the accoucheur
- (3) Absence of deleterious effects on both mother and child

The combination of nembutal and chloral seems to fulfil these criteria well and to possess other advantages in addition

The following account is based on a small series of cases numbering 30 in all

### DESCRIPTION

Nembutal is one of the heavy series of barbiturates whose main characteristic is a preponderance of hypnotic over anæsthetic action

Nembutal was introduced into this country some six years ago. It has since been widely used and become well known as a basal narcotic or rather amnesic, and also as a hypnotic and sedative. It is commonly used in gelatin capsules containing  $1\frac{1}{2}$  grs of the powder

### METHOD

The method of administration about to be described has been on similar lines to that used by O'Sullivan and Craner<sup>1</sup> in their series of cases

*Time of commencing narcosis* This varies according to whether the patient is a primipara or a multipara

Primiparous patients are instructed to complain when the pain they are experiencing is becoming too severe to be easily tolerated, and the first dose is then given, having due regard to the psychology of the patient as to whether the pain is really as described, and also endeavouring not to start the narcosis much before two-fifths to three-fifths dilatation of the cervix

In dealing with multiparous patients it is a good plan to try to obtain a history of the duration of previous labours and thus to estimate the probable duration of the one under treatment. If the latter is likely to be short, an endeavour should be made to start the narcosis not less than three

hours before the expected delivery, and if possible or necessary earlier. In other cases, where the labour is likely to be longer, a start may be made as before when the patient makes her complaint, and if possible not earlier than two-fifths dilatation.

The first dose consists of nembutal grs 3 and chloral grs 30. The second, about one and a half hours later, consists of nembutal grs  $1\frac{1}{2}$  and chloral grs 20-30. Subsequent doses of nembutal grs  $1\frac{1}{2}$  and chloral 20-30 are given every two to three hours, the length of time between doses being best gauged by experience. The well-known "memory test" may be of some help in this connexion.

*Total dosage* Should not exceed, or greatly exceed nembutal grs  $7\frac{1}{2}$  and chloral grs 120 in eight to ten hours. Only in exceptional cases is a longer period of narcosis necessary and in these the administrator's discretion and experience and the condition of the patient must determine the giving of additional amounts.

The following points in the administration are important.

(1) The patient's stomach should, if possible, be empty before giving the first dose and only fluids given thereafter.

(2) The dose of nembutal should always precede the dose of chloral by about ten minutes. Giving both together tends to excite nausea and possibly the dose may be vomited to the great confusion of the administrator, who will naturally not be able to estimate how much of the drug may or may not have been retained.

(3) A quarter of a teaspoonful of bicarbonate of soda in a little water to wash down the capsule of nembutal may be given. This will assist rapid absorption as barbiturate drugs require a considerable amount of alkali for solution in the body fluids. Further, an acid reaction in the stomach militates against regular and steady absorption.

(4) Pricking the capsules with a needle before administration is another simple device which will assist in promoting a rapid absorption of the drug.

(5) Giving the nembutal in solution or as a powder unfortunately tends to provoke nausea and vomiting owing to its unpleasant taste, which is extremely difficult if not impossible effectually to mask.

The dose of chloral is best given in a sweetened and flavoured draught such as

R

Chloral	gr $\text{v}$
Ol Auranti	℥ii
Syr Simplex	3i
Aq Chlorof	ad $3\frac{1}{2}$

After the first dose has been given the patient should take to bed and lie down and try to go to sleep. The lights should be shaded and all necessary noise suppressed.

#### PERMISSIBLE ADJUVANTS

(1) Nitrous oxide and oxygen, or a little ether may sometimes be necessary for the actual crowning and delivery.

A small addition of ether may also be an advantage at times to control too rapid or forcible contractions, which might result in damage to soft tissues. Usually a few whiffs only are necessary, never enough to produce any degree of acidosis post-partum, with its unpleasant sequelæ of nausea and vomiting.

If any obstetric operation has to be carried out, good and adequate surgical anæsthesia is secured with greatly reduced quantities of inhalation drugs, and with it a consequent freedom from post-anæsthetic troubles.

#### NON-PERMISSIBLE ADJUVANTS

*Morphia and hyoscine* should be avoided at any stage. The danger of foetal asphyxia with these powerful depressants is very real especially if used in conjunction with nembutal when a considerable degree of synergistic action results, and their employment should never be necessary. Irving, Berman, and Nelson,<sup>2</sup> using scopolamine grs 1/100-1/150, in addition to nembutal, repeated in some cases, reported 86 per cent of perfect amnesias in the mothers, but at the same time only 63 per cent of their babies breathed spontaneously at birth. McGuiness,<sup>3</sup> in his series of cases, abandoned the addition of morphia by reason of the frequency of oligopnoea as a foetal complica-



tion, although it had a marked effect on the mothers. In the only two cases of the present series receiving morphia and/or scopolamine, both of which were in all other respects perfectly normal and without difficulty for the accoucheur, white asphyxia nearly produced a tragedy (*qv*). No other case gave rise to any anxiety on this score.

*Chloroform* It seems doubtful whether such a powerful protoplasmic poison should be employed in view of the many other less toxic anæsthetic agents of great efficiency to-day available. It is likely to prove a peculiarly dangerous drug in combination with a barbiturate.

*Intravenous barbiturates* are not desirable owing to the very real danger of cumulative effects.

*Paraldehyde* offers no theoretical objections, but it should not be necessary in the earlier stages of labour and it is difficult to see how it could be of much use at the time of delivery.

*Avertin* The same applies to this drug with the additional observations that the combination of avertin with a barbiturate is not a generally favoured one and that the former definitely tends to slow down the progress of the labour.

### CLINICAL EFFECTS

About half an hour elapses before the first dose begins to exert effective action. The maximum effect should be manifest in about one to one and a half hours.

The patient sleeps between contractions, breathing with some degree of stertor. She can be roused by loud speech and made to take fluids and glucose. The conjunctival reflex is usually sluggish or absent, the corneal reflex should always be present.

During contractions the patient groans and frequently complains of suffering and the inadequacy of relief afforded her but can converse and is co-operative. She will obey instructions and can be reasoned with.

Sometimes there is some restlessness but it is easily controlled, the chloral exerting a definite effect in this direction. The patient's colour remains good and general appearance normal except for heaviness of the eyelids and a sleepy

confused look. Speech is often rather drunken and slurred and dimness of vision and diplopia is sometimes complained of.

After delivery the patient will usually sleep peacefully for some hours and when she awakes amnesia is found to be excellent.

Often a mother after delivery will ask to be shown her child, and shortly after being shown it will repeat the request, perhaps several times, convinced on each occasion that she has not yet seen the infant, thus showing that the influence of the drug has really reduced her memory time to a very short period.

### PHYSIOLOGICAL EFFECTS

**MOTHER** *The pulse* remains normal. No effects have been observed on the heart.

*The lungs* are not affected in normal patients. One or two patients have been slightly "chesty" for a few days after delivery, but in each case were the type that is prone to bronchitis and had coughs before labour started. The only patient who gave rise to any anxiety in respect of the respiratory system was one who developed a pleural rub, but this was readily attributable to errors of room ventilation, and cleared up in a few days without any untoward results subsequently.

*The alimentary tract* Nausea and vomiting are very rare if attention is paid to the technique of administration. No other effects on the system have been noted during the puerperium.

*Liver and kidneys* No harmful effects seen.

*Labour* is quite certainly not prolonged though it is doubtful if it is actually accelerated as has been suggested. This latter point is admittedly very difficult to assess. The length and strength of the contractions do not seem to be affected, nor do the intervals between them appear to be altered after commencing the narcosis.

The method does not seem to increase the amount of haemorrhage, although this again is a very difficult factor to assess.

**FÆTUS** In no case receiving nembital and chloral alone has there been any difficulty with the babies at birth. All have cried well on delivery and thereafter behaved normally.

## RESULTS

These have been encouraging. An attempt has been made to summarise and classify them in the following way.

Those patients who have had no memory of their labour after the start of the narcosis have been classified as "excellent". Those who remember isolated incidents as in a nightmare are grouped as "good". While those who claim to have remembered a good deal of their labour in a confused way but admit to little actual pain or suffering are labelled "moderate". The description "failure" has been applied to those who report that they had little benefit from the drugs.

Of the whole series the results in round figures were Excellent, 50 per cent, good, 31 per cent, moderate, 16 per cent, and failure, 3 per cent.

## ILLUSTRATIVE CASES

The following selection of cases from the series illustrates the method and indicates the course of the narcosis —

(1) Mrs B, aged 25 years, primipara. Very neurotic and self-centred young woman. History of chronic bronchitis. Chest N A D on examination. Membranes ruptured early at 6 p.m. At 10.50 p.m. first dose given. Nembital (N) 3 grains, chloral (C) 30 grains. Dilatation two-fifths. Rather restless, complained of pain and grumbling continuously. Apparently no effect. 12 m.n., N  $1\frac{1}{2}$ , C 30. 1.50 a.m. very restless and complaining querulously, morphia  $\frac{1}{8}$  grain. 3.40 a.m. N  $1\frac{1}{2}$ , C 30. 6 a.m. N  $1\frac{1}{2}$ . 6.30 a.m. C 30. 7.30 a.m. a little ether for forceps application by accoucheur. Baby born in severe white asphyxia, and only resuscitated by mouth to mouth breathing after all other measures, including coramine, CO<sub>2</sub>, etc., had proved of no avail. Amnesia complete. Post-partum hæmorrhage, 30 oz. Subsequent transfusion of 600 c.c. of

citrated blood. The mother, who never ceased asking questions about herself and imagining her condition to be abnormal in every possible way, developed pyrexia within four hours of delivery which gradually rose to 101 degrees without any physical signs except slight bronchitis. The pyrexia subsided on the fifth day within a few hours of being seen by a physician, who reassured her, on her anxious and repeated request. Psychological (?)

(2) Mrs H, 25 years of age, primipara. First dose at 3/5th dilatation. Total dosage Nembutal 6 grams, chloral 40 grains. Difficult labour. Duration of narcosis 6 hours. Ether (for forceps) at end only. Foetus, spinæ bifida and some degree of hydrocephalus. Baby born alive and breathing normally. Patient slept six hours after delivery and had complete amnesia from the time of commencing the drugs. Developed a pleural rub. Normal after a day or two. No after-effects.

(3) Mrs S, 28 years of age. Primipara. First dose given late in second stage three hours before delivery. Total dosage, N 4½, C 40. A few whiffs of ether at delivery. Mother and foetus perfectly normal. Complications, nil. Amnesia complete.

(4) Mrs W, 21 years of age. Primipara. First dose on complaining of pain. This was given as nembutal in solution. Patient was nauseated and shortly after vomited. It was considered that practically all the drugs had been lost and omnopon ½ gr and scopolamine 1/150 gr were injected. Patient delivered six hours later. Amnesia complete but baby severely narcotised and resuscitated with some difficulty.

(5) Miss S, 28 years of age. Primipara. A wildly hysterical and uncontrolled person of low intelligence. Struggled and screamed from the start of labour. Total dosage, N 4½, C 40. Duration nine hours. Fought and struggled most of the time. Normal delivery. Baby normal. Insisted no amnesia produced.

(6) Mrs J, 30 years of age. Multipara. Four previous labours, all rapid. Total dosage N 3, C 30. Given at start of labour. Delivered in two and a half hours. No ether. Complete amnesia.

(7) Mrs V, 23 years of age Primipara First dose on complaint of pain Total dosage N 6, C 90 Duration of narcosis, 10 hours A few whiffs of ether at crowning Mother and baby very satisfactory Amnesia good Remembered isolated incidents as in a nightmare No pain

(8) Mrs B, 26 years of age Primipara Total dosage N 6, C 90 First dose on complaining Duration narcosis nine hours No ether Mother and baby satisfactory Amnesia complete

(9) Mrs P, 31 years of age Primipara Very nervous, but courageous Total dosage N 4½, C 60 Duration, eight hours Amnesia only moderate The patient reported that she felt as if in a nightmare Felt a little pain but suffering not great Dosage obviously insufficient

(10) Mrs B, 33 years of age Multipara Four previous labours Total dosage N 6, C 90 A little ether at end Duration, 10 hours Amnesia good Patient reported her labour as the most comfortable she had ever had Remembered occasional incidents in a dreamy way No actual suffering Dosage not quite sufficient otherwise amnesia would have been complete

#### ADVANTAGES OF THE METHOD

*Mother* (1) Labour is not prolonged and there is no evidence to show that complications are increased (2) There are no unpleasant after-effects and no subsequent "hangovers" The patients feel quite comfortable after waking from their post-partum sleep (3) Amnesia is aimed at, and not periods of semi-anæsthesia alternating with periods of consciousness

*Foetus* (1) No dangers of respiratory failure at or after delivery (2) Absence of prolongation of labour materially decreases the danger of damage to the baby

*Obstetrician* (1) Simplicity is an outstanding characteristic of the method (2) The drugs are carried in a couple of small bottles and thus there is no cumbersome apparatus to be transported (3) The cost is but a shilling or so Compare this with hours of gas or oxygen, and the upkeep of the necessary apparatus to administer it (4) The obstetrician can count on a really co-operative patient

## DISADVANTAGES

These are few. There is frequently some degree of restlessness, but it is not excessive and is always easily controlled, the patient usually displaying a gratifying degree of docility.

## CONTRA-INDICATIONS

*Kidneys* Elimination of the end-products of the metabolism of nembutal being via the kidneys, care should be taken if these organs are not up to normal function.

*Liver* Damage to or disease of the liver is a well-known and understood contra-indication to the barbiturate group of drugs, as it is in the liver that they are detoxicated. Nembutal is no exception to this rule.

*Toxæmias of pregnancy* As these complications involve damage to the above-mentioned organs, they should also be regarded as contra-indications.

*Lungs* Bronchitic and "chesty" patients are apt to have their condition aggravated by the administration of nembutal owing to a certain amount of depression of the respiratory system. Patients having any degree of respiratory obstruction are also not good cases for the use of nembutal.

*Heart disease* Advanced cases are naturally very difficult to handle whatever methods are adopted, and require very careful observation and treatment. Whilst the direct effects of nembutal on the heart are negligible the associated respiratory depression may further embarrass an already over-taxed organ.

In cases of cardiac disease where compensation is good there is no contra-indication, rather the reverse. Because if the patient has to bear great suffering, it can only increase the amount of shock and exhaustion which her system will be called upon to bear.

## COMPARISON WITH OTHER METHODS

Almost every drug having a claim to amnesic, analgesic or anæsthetic properties has been used to relieve suffering in labour at one time or another. To attempt to catalogue

all these would be tedious and without profit. Mention, therefore, will only be made of some of the more outstanding and successful ones.

*The classical twilight sleep* is one of the best-known methods dating back to the beginning of this century. Although very pleasant and effective from the point of view of the mother, its conduct requires a good deal of skill and experience and the powerful drugs used, morphia and hyoscine, have their own peculiar dangers to the foetus, which have been emphasised here and are well known to all. The method is one which definitely slows labour.<sup>4</sup>

*Chloroform* Has the distinction of being time-honoured. It is also a highly toxic drug and a powerful protoplasmic poison, however, and in spite of the oft-made assertion that women in labour "tolerate" it well (for which there seems to be no scientific foundation) it has been responsible for a good many tragedies, often not recognised as being referable to the drug. Post-partum liver poisoning is an example which has been described.<sup>5</sup>

*Gas and oxygen* As an anæsthetic in labour, although almost ideal from its own point of view, i.e., that of complete unconsciousness of the labour with absence of deleterious effects, does not allow of a co-operative patient, and is, in addition, a very expensive method given over long periods requiring the whole-time presence of an anæsthetist.

*Gas and air analgesia* is still on trial, and whilst possessing undoubted advantages does not seem to give a sufficient degree of analgesia to a fair proportion of the patients.

*Avertin* has been used to a certain extent but the contractions are apt to be adversely affected and thus the labour tends to be prolonged.<sup>6</sup> Also rectal injection is not a manoeuvre undertaken with ease and success during labour, at least in its later stages. Oligopnoea in the foetus is also apt to occur.<sup>7</sup>

*Intravenous barbiturates* The "light" barbiturates used in surgical anaesthesia produce too short an effect and result of course in complete consciousness, while the "heavy" members of the group are not altogether safe drugs for intravenous administration.

## CONCLUSIONS

In nembutal used in conjunction with chloral hydrate we have a method of relieving suffering in childbirth which goes a long way towards meeting the above disadvantages.

(1) It is not toxic, safe for the mother, and does not affect the foetus

(2) Labour is not prologed, and the complications are not apparently increased

(3) It is cheap and easily transportable

(4) It is simple to administer

(5) Amnesia plus a high degree of co-operation result

(6) It is effective over relatively long periods

(7) After-effects are conspicuous by their absence.

(8) Whilst no case has given the least cause for alarm referable to the action of the drugs, it is definitely dangerous to add morphia or hyoscine

(9) The doses employed up to the present have been on the cautious side and it is felt that with further experience slightly larger doses or shorter intervals would improve the results. On the other hand, the use of big doses such as nembutal 6 grains, initially followed by 3 grains in three hours, and then 1½ grains every two hours which have been described would seem to be rather heroic and not without risk. They cannot be recommended

(10) Lastly, the portability, simplicity, and ease of administration give the drug a special degree of suitability for single-handed general practitioner midwifery

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## THE HICKMAN MEDAL

This medal, as many of our readers are doubtless aware, is awarded every three years by the Council of the Royal Society of Medicine on the recommendation of the Council of the Section of Anæsthetics. This year's award is the second to be made. The recipient is Dr I W Magill of London, Senior Anæsthetist to the Westminster Hospital. The only previous award was to Dr Wesley Bourne of Toronto. We congratulate Dr Magill on the honour which he receives, and are no doubt voicing the general opinion of anæsthetists when we state our belief that no one in Great Britain more fully deserves the medal than he does. Magill's work in perfecting the technique of endotracheal anæsthesia, in popularizing this method, and in adapting it to the demands of severe lung surgery would entitle him to distinction even apart from much other fine clinical work which lies to his credit.

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## THE DIPLOMA IN ANÆSTHETICS

Since our last issue the D A has been conferred on the following practitioners —

W E Brown, A H Diamond, Hugh Hunter, Kathleen McLyon, Kathleen M Oldham and E S Rowbotham

## THREE DISTURBING CASES OF SPINAL ANÆSTHESIA ADMINISTERED ACCORDING TO THE ETHERINGTON WILSON TECHNIQUE

By C. W. H. VAN DER POST, B.A., M.R.C.S.E., L.R.C.P.

THE advantages of the Etherington Wilson technique for administering spinal anaesthetics are well known and may be summarized as follows

(1) Ease of performing the lumbar puncture due to the sitting position,

(2) Comfort to the patient in avoiding a forced hyperextension over neck and round the knees and in avoiding too the turning of the patient on the face and again on the back,

(3) Easy control of the level of anaesthesia by the diffusion-rate of the anaesthetic fluid used,

(4) The quickness with which the patient can be got ready for the operation, an advantage in emergency work or otherwise when time is pressing,

(5) The use of a drug, which in the concentration of 1/1500 is decidedly less toxic than numerous of the novocaine derivatives, and one, too, which does not contain any alcohol

Bearing in mind these advantages, this technique was employed in the following three cases, which have rather badly disturbed my confidence in the Etherington Wilson technique. But before further discussing this disturbance let us relate the three cases

### Case One

A woman of 29, on whom Cæsarean section was to be performed. She was thin and very sallow in complexion and looked rather ill. On examination, nothing could be detected to be wrong. Her B.P. was 118/70, chest and heart were perfectly normal, she had a steady

and regular pulse of 60, and the urine showed no abnormality at all. Premedication was  $1/3$  omnopon and  $1/150$  scopolamine  $1\frac{1}{2}$  hours before operation. Ten c.c. of  $1/1500$  percarine were injected in the third lumbar space, taking 20 seconds over the injection, and the patient was sat up for exactly 20 seconds, and then  $1\frac{1}{2}$  grains of ephedrine were given hypodermically, as soon as she had been put in a Trendelenburg position. The operation was started about ten minutes after completion of the spinal, and the patient's breathing and pulse were quite good until the surgeon commenced to sew up about twenty minutes after the initial incision and thirty minutes after completion of the spinal, when the patient exhibited decided signs of air hunger and the radial pulse was hardly perceptible. The Trendelenburg was at once increased to a steep angle, 5 c.c. coramine and  $3/20$  grain of lobeline were given hypodermically and the continuous flow of oxygen was greatly increased and a little carbon dioxide given as well. For about ten minutes the patient gave us great anxiety until she was at rest in bed, when the regular respiration and the improvement in the pulse gave assurance of the patient's safety.

Though this case does not sound as dramatic as it really was, the patient very nearly died, but it was not as bad as the next two cases, both of these patients having died.

#### *Case Two*

A native woman of about 33 years of age, B.P. of 130/95, good pulse, normal chest and urine, having been in labour for about 15 hours. A spinal anaesthetic was administered exactly similar to that in the first case and this was completed at 5.25 p.m. The operation of Caesarean section was started at 5.30 p.m., and at 5.57 p.m., when the surgeon started to sew up, the anaesthetist asked leave to be excused on account of another pressing case. During the operation the patient had given no cause for alarm whatsoever, and having assured both himself and the surgeon that the patient's condition was good, the anaesthetist departed. The operation was completed successfully at 6.10 p.m., and at the conclusion the resident house surgeon felt the pulse and also assured himself that the patient's condition was satisfactory. He accompanied the surgeon to his car outside and received an urgent call to return to the theatre, where on his arrival he found the patient dead on the Trendelenburg trolley.

It is possible that this patient was upset by rough handling during her removal from the operating table to the spinal trolley, but the sister in charge is most efficient and has assured me that that was not the case.

*Case Three*

This too was a native woman of about 35 years of age, somewhat stout, with a B P of 125/90, a full normal pulse of 84, and no anaemia as far as could be judged from her conjunctivæ or mucous membrane of the mouth and lips. The diagnosis was uncertain, but a laparotomy below the umbilicus was to be performed. Ten c.c. of pericaine were injected into the third lumbar space and the patient sat up for only 15 seconds. An immediate Trendelenburg position was assumed and 1½ grains of ephedrine injected and a free flow of oxygen given all the time, as in both of the previous cases. The spinal was completed by 2.20 p.m. and the operation started at 2.30 p.m. The pulse was good, but at about 2.35 p.m. the patient's alæ nasi were functioning to excess and respiration was irregular and sighing in character. A little carbon dioxide and a steeper Trendelenburg produced a poor response on the part of the patient and the carbon dioxide was discontinued at 2.38 p.m. Breathing was fairly good for a few minutes and the pulse quite satisfactory. The surgeon found an ectopic and began to close the abdomen at 2.40 p.m., and soon after the patient's breathing ceased and the pulse was hardly perceptible. Carbon dioxide now produced no response, and a series of dramatic restorative measures were executed. Five c.c. coramine into the arm without response, 3 c.c. coramine into a vein and massaged to the heart without response, 5 c.c. coramine straight into the heart through the third right interspace without response, adrenaline straight into the heart without response, cardiac massage and another 5 c.c. into the left ventricle and some more adrenaline straight into the heart all had no response, and the case was abandoned at 3 p.m., artificial respiration having been performed continuously since 2.45 p.m.

*Autopsy*

In both these last two cases the post-mortems revealed nothing abnormal whatsoever, apart from the usual asphyxial symptoms.

*Verdict*

The second case presumably died from respiratory failure, and the third probably from both respiratory and cardiac failure, as the heart was felt to be extremely flabby on performing cardiac massage.

Now Dr Jarman says in his article on "The Combination of Intravenous and Spinal Anæsthesia, using Pentothal and Pericaine" that "there is probably a considerable margin of safety even after 80 seconds" of sitting the patient up, and Preissecker of Vienna writes in his "Lumbalanasthesie in

der Geburtshilfe und Gynakologie " that the patients (10 cases) " were allowed to sit up for one to two minutes and only then were put down again, all these ten cases were fully anaesthetized and only in one case did harsh vomiting make its appearance " One would conclude that it is quite safe to sit them up for any length of time, for Preissecker thinks that the anaesthetic fluid is so rapidly absorbed by the roots that when it reaches the fifth dorsal level there is no more fluid left to be absorbed If this is true it would appear that there was some definite contra-indication to a spinal anaesthetic in the above cases, more especially in Nos 2 and 3, but there was no such obvious contra-indication

In all three cases anaesthesia was perfect at least as far as the umbilicus, and since these unfortunate occurrences as little as 8 and even 7 c c of 1/1500 percaïne have been used, sitting the patients up for only 15 seconds, and in each case anaesthesia has been adequate for a Cæsarean section

It would appear to me that the diffusion rate of any anaesthetic solution in the spinal column is not as constant as Etherington Wilson assumes He has worked out his theory on the basis of the assumption that the specific gravity of the cerebrospinal fluid is 1.008 Now the specific gravity has been stated to vary as much as from 1.003 to 1.020, and in addition the pressure to vary from 50 to 150 m m of water Is it not conceivable that the diffusion rate of 1/1500 percaïne of a constant specific gravity of 1.003 may vary considerably according to the actual specific gravity of the C S F, the pressure of the C S F, the amount of percaïne injected, and even the rate of injection?

At the time the above spinal injections were administered it did not occur to me to ask the patient whether she was menstruating or whether she had just passed through her menses, but it would be interesting to hear from one of your readers whether the menstrual flow, or any hæmorrhage for that matter, affects the specific gravity of the C S F If it does and it also lowers the pressure of the C S F, as it must do by the general lowering of the physical activity and tone of the patient, it seems fairly certain that the rate of diffusion 1/1500 percaïne solution will be materially altered thereby And from this point of view it would be interesting too to

hear whether the effects of spinal anæsthetics are more constant and more easily controlled in male than in female patients

I shall be extremely gratified if any of your expert readers could give me definite information about these difficulties, and especially if they could refer me to any text-book or treatise with exact information on these points. Both Case No. 1 and Case No. 2 were patients on whom an ordinary general anæsthetic such as chloroform, ether, or a *c*-and *c*-mixture, an intravenous anæsthetic or gas and oxygen would have permitted the surgeon to complete his work with reasonable ease and with almost absolute safety to the life of the patient there can hardly be any doubt about that, and in retrospect it seems an unnecessarily cruel and inhuman act to have submitted them to the greater danger of a spinal anæsthetic, were it not that the anæsthetist believed that a spinal, especially a low spinal, in these cases was just as safe. Though there was no difficulty experienced in performing the lumbar puncture, and though exceptional care was taken to make the anæsthetic as safe as was possible, it would appear that the judgment of the anæsthetist was at fault or else that the safety factor of spinal anæsthesia or of percaine as a spinal anæsthetic is smaller than is generally believed. It would be interesting and most helpful to me to receive a candid criticism of my work in these cases and/or to be put wise to any misconceptions under which I may have laboured.

## CARBONIZED ETHER FOR ANÆSTHESIA

By DR K E MADAN, M D , D O M S , F I C A

*Lecturer in Anæsthesia, K E Medical College, Lahore*

**D**URING the course of a prolonged operation under "open" ether anæsthesia, it is sometimes found that there occur irregularities in breathing, e g shallow breathing, and temporary periods of apnoea, as a result of alkalosis. These periods of apnoea may last even for a couple of minutes in cases where oxygen has been allowed to bubble through ether, in such cases on administration of  $\text{CO}_2$  the breathing is resumed, showing that acapnia is the cause of apnoea. Sometimes when much oxygen is continually allowed to bubble through ether for about an hour or more, these periods of apnoea recur, particularly when morphia has been injected for premedication, in doses higher than gr  $\frac{1}{6}$ , i e gr  $\frac{1}{4}$ . The administration of  $\text{CO}_2$  to be effective when there is complete apnoea, should be accompanied by compression of the chest and rhythmic traction on the tongue during inspiration, i e some sort of artificial respiration. The onset of apnoea is preceded by slow and shallow breathing, and that is the proper time when  $\text{CO}_2$  should be administered to stimulate the respiratory centre. To get rid of these drawbacks of open ether, which are met with in some cases, such as irregularities in breathing and apnoea, I have, for some time, been previously carbonizing the ether before it is used by the open method. This was done by allowing a good rapid stream of  $\text{CO}_2$  gas to enter for not more than two minutes a one-pound bottle, filled three-fourths with pure anæsthetic ether. A long metal tube through the cork of the bottle and bent outwards may be used, and the rubber tubing from  $\text{CO}_2$  cylinder can be connected to it. The bottle is then at once corked and shaken. Specimens of

ether so treated were examined by the Government Chemical Analyser and reported to be quite safe for anaesthetic purposes

I made use of this carbonized ether by the open method in a large number of abdominal and other operations and was struck by the fact that even in lengthy operations the breathing in all cases was deep, regular, and uniform and that there was less secretion in the mouth and the incidence of post-anæsthetic nausea and vomiting was definitely reduced. Neither post-operative bronchitis nor pneumonia was seen in any of these cases. The pupils remained contracted, the pulse was of good volume and tension and generally not over 100, and the respirations under 30, the colour and capillary circulation remained good. I found that the use of carbonized ether is useful in operations where shock is present or anticipated, because the increased and regular depth of respiration helps the circulation, and so as the tone of the vaso-motor centre is maintained, the blood-pressure is kept up. In gall-bladder cases where a bridge is put under the back, and in kidney and other cases where the patient is kept turned on one side, sometimes the breathing is embarrassed, and in other cases where there is already some dyspnoea, the use of carbonized ether avoids laryngeal spasm and is safe and satisfactory. A gentle stream of oxygen was often allowed under the mask and found advantageous. The amount of carbonized ether required to maintain deep anæsthesia with good relaxation was found to be definitely less than with ordinary ether as used by the open method. The use of the author's double mask is specially advantageous and economical as it reduces the loss of ether by evaporation, and so less ether is required, and consequently there is less shock, and toxic effects of ether are lessened.

In kidney operations, e.g. nephrectomy, this is a safe procedure, for if there is any hypofunction of the other kidney, as little ether as possible should be used to avoid the cessation of kidney secretion, and carbonized ether is a stable and less toxic anæsthetic. In places where open ether is generally used, and in operating theatres in which cylinders are not allowed to remain handy near the anæsthetist, ether for want of space or lessening the congestion of the



equipment there, this procedure of carbonizing the ether beforehand will be found useful and satisfactory, and will relieve the anæsthetist of the worries due to irregular breathing or periods of apnoea. Carbonized ether is also useful by the endotracheal route.

I have tested this method for the last six months, in almost all sorts of cases, e.g. in jaundiced patients, those in toxic states, and in state of surgical shock, and have not come across any harmful effects, nor were any twitchings or convulsions ever seen with this carbonized ether even in very hot weather, though with ordinary ether I have several times seen the typical late ether convulsions in prolonged cases. In a number of cases, I filled this carbonized ether in the ether container of Shipway's apparatus and allowed oxygen to bubble through it as required, and no harmful results were noticed, and even ether so treated was reported by the chemical analyser to be fit for anæsthetic purposes.

In view of the above-mentioned advantages I advocate the use of carbonized ether for open method, particularly in cases where breathing is irregular, and this suggestion may be taken up by the ether manufacturers, who can treat the ether as stated above before putting it on the market. However, it must be emphasized that the ether should be mixed with only the minimum amount of  $\text{CO}_2$ , which is just sufficient to maintain the depth of breathing. If the rate of breathing is increased, the use of carbonized ether should be stopped for the time being, as there may be undue accumulation of  $\text{CO}_2$  in the system.

## ABSTRACTS

*"Post-anæsthetic pulmonary complications"* L. E. OSBORNE in *Brit Med Journ.*, February 5th, 1937, p 279

THE author declares "post-anæsthetic" to be a misnomer and that general anæsthesia by itself is of very little importance in the ætiology of post-operative lung complications. The typical post-anæsthetic pneumonia is the result of many minute pulmonary emboli from the operation, etc. and so is slightly more common after local than after general anæsthesia. Its most important causes are trauma, mobility of the affected part and sepsis. It is further pointed out that aspiration of foreign matter, blood and mucus for example, in the trachea and lungs is more easily effected than is commonly supposed, and occurs with disastrous result even in the absence of any anæsthetic. The author gives interesting cases illustrating his views, including one in which a portion of tooth inhaled while the patient was about her ordinary avocations caused fatal pulmonary oedema. He believes that embolism and aspiration are both important causes of post-operative lung lesions though neither alone can explain all cases.

In the same issue, on p 283, is an interesting account of a tumour of the vocal cord following on endotracheal anæsthesia. The tube had been passed nasally "after repeated attempts"

*"Effect of anoxia on the action of nitrous oxide in the human subject"* J. H. BENNETT and M. H. SLEVERS in *Journ Pharm and Exper Therap*, December 1937, p 329

THE authors point out that anoxia is a necessary accompaniment of deep anæsthesia from nitrous oxide in any subject

who has not received previous medication by a sedative or hypnotic drug. They carried out a number of experiments to find out at what level anoxia contributes to the depressant action of  $N_2O$  and whether anoxia elevates the sensory threshold. The chief practical outcome of their investigation is the conclusion arrived at that there is probably a causal relation between anoxia and post-anæsthetic sickness, and this is quite in accordance with clinical results of nitrous oxide administration, although clinicians have generally attributed the sickness to excess of  $CO_2$  rather than to lack of oxygen.

*"Gas-oxygen and cerebral congestion"* H J BRENNAN in *Lancet*, February 5th, 1938, p 315

THE question is discussed chiefly in connexion with operations on the brain. Three causes are assigned for congestion during anæsthesia by gas-oxygen, viz

- (1) An insufficient proportion of oxygen in the mixture
- (2) Respiratory obstruction
- (3) Faulty position of the patient

The author recommends giving omnopon-scopolamine an hour before avertin of which three-quarters of the full estimated dose is injected during about a quarter of an hour. No atropine is given. Usually it is possible to give 20 per cent oxygen with the nitrous oxide after this premedication. The introduction of a wide-bore tracheal tube is the first and essential step to completely smooth and unobstructed respiration. The author does not find that spraying the pharynx with cocaine prevents reflex coughing, because this is due to the distal end of the tube rubbing up and down on the wall of the trachea. Efficient local anæsthesia of the part of the trachea thus affected is what is needed. After much trial an effective lubricant was found

paraff dur	gr 15
cera alb	gr 30
paraff moll alb	1 oz
percarne base	10 per cent



before the endotracheal injection to the point of light sleep. It is interesting to note that for pelvic operations on ward patients open ether is mostly used, chiefly because these patients are most suitable for teaching purposes, because of the comparative freedom from post-operative respiratory and other complications. For brain surgery endotracheal ether through a large catheter and from a motor-driven vaporizer has become the choice. For inducing anaesthetic without excitement chloroform is introduced when necessary. Thyroid surgery is conducted mostly under nitrous oxide, preliminary sedatives to ensure quiet being given. Spinal analgesia with novocaine is used for lobectomy of middle and lower lobes. The good results that Shields and colleagues have achieved in this matter are well known and have been imitated to some extent in this country.

In association with the above readers are referred to a note on "Anaesthesia for upper abdominal surgery" (*Lancet*, February 12, 1938, p. 386) in which there is some account of the paper (H. K. Ashworth) and discussion on this subject at the Royal Society of Medicine.

*"Physiological phenomena related to anaesthesia"* C. F. SCHMIDT in *Anaesthesia and Analgesia*, January-February 1938, p. 29.

THE author deals chiefly with physiological and chemical actions which affect the respiratory centre. An interesting part of his paper concerns the carotid sinus or carotid body, small objects to which only comparatively recently have been assigned important influence on respiration. It is now known that reflexes are aroused in specialized receptors in the carotids by changes in the chemical composition of the blood and that breathing can be strongly stimulated in this way. These reflexes constitute an important part of the body's defence against anoxaemia and high carbon dioxide tensions. These chemically aroused reflexes are distinct from those elicited in the carotid sinus by changes in arterial pressure, the latter having important

bearing on control of the heart rate of different parts of the body has little on respiration. The carotid receptors on the other hand, which are responsive to chemical changes in the blood, have an effect on circulation though a powerful effect on respiration. The carotid body appears to be an organ in which a transformation of chemical change in the blood into afferent nerve impulses which are sent to the respiratory centre, it is the first of the "chemoreceptors". "chromaffin tissue", which are scattered throughout the body in relation to major arteries, for which a definite function has been made out.

"Minor points in local anaesthesia" P. D. Wood, *Canad Med Assoc Journ*, March 1958, p. 216

THE author believes in effective dosing beforehand with pain-relieving drugs, such as morphine and not with large amounts of those designed to allay apprehension, barbiturates for example, since these may lead to delirium and uncontrollability. Small doses of barbiturate he approves of because they protect against the convulsive effect of intravenous injection of local anaesthetics. If the metabolic rate of the patient is high responsiveness to painful stimuli will be exaggerated. In these circumstances the doses of all drugs used should be increased, whereas they should be reduced in infancy, old age, anaemia, myædæmia, or weakness from any cause.

He believes that most cases of toxicity from local anaesthesia are in reality not from the anaesthetic drug but from excessive amounts of epinephrin used for vaso-constriction. If very small amounts of the latter are used and the injection made slowly no trouble occurs. The technique of injection is described in detail. The use of metycaine instead of novocain appears to make failure less frequent.

## CORRESPONDENCE

*To the Editor of the British Journal of Anæsthesia*

## STOPPAGE OF BREATHING DUE TO LARYNGEAL SPASM

Dear Sir,—In a recent number you suggested that anæsthetists might discuss anæsthetic deaths in the journal. During the past year I have had four cases which have not been very successful.

(1) A man aged 75, operation, prostatectomy. An attempt was made by the surgeon to administer a spinal anæsthetic, but the patient had very marked arthritis of his spine and the spinal anæsthetic was not successful, so I was asked to administer an inhalation anæsthetic. I gave the patient gas-oxygen-ether from a Boyle's apparatus and no trouble was experienced during the induction. When the surgeon passed his finger into the bladder to remove the prostate, however, the patient developed severe laryngeal spasm and stopped breathing. Artificial respiration was tried, but without avail, and the patient's condition became extremely grave. An endotracheal tube was then introduced and oxygen administered through this. The patient's condition failed to improve and cardiac massage was started. The patient gradually recovered, but died twenty-four hours later without recovering consciousness.

(2) A man aged 86, with acute retention of the urine. He was a very ill man on admission, and gas and oxygen was administered with a view to suprapubic puncture. Immediately this was done the patient stopped breathing, due to laryngeal spasm. Artificial respiration again failed, and then an endotracheal tube was passed with some difficulty and oxygen administered. The patient gradually recovered, but died the next day. He was found to have a large carcinoma of the bladder.

(3) A man aged 75, admitted with a urinary calculus. Gas and oxygen was administered with drainage. The patient stopped breathing. The trachea was opened, and only recovered after the trachea was intubated with an endotracheal tube and the administration of oxygen. The patient made good recovery.

(4) A man aged 75, suffering from a large inguinal hernia. Gas and oxygen was administered. About five minutes the patient stopped breathing. The trachea was opened in spasm. An endotracheal tube was inserted and oxygen was administered, but the patient was not revived. The trachea was intubated and the patient was induced to breathe again. The operation was completed and the patient recovered.

Now there was a marked resemblance between the two patients. All were elderly men, all were of the red-faced type, all had bushy eyebrows, all had large chests, all had hard, rigid chests which made inspiration almost impossible. All four patients had been breathing for apparently the same cause—laryngeal spasm—and they stopped breathing to a degree which produced a very serious alarm.

As a general practitioner anaesthetist I should be infinitely obliged if some anaesthetist of wide experience would tell me how to overcome these difficulties, or how to avoid them in the future.

Yours faithfully,  
G P

*To the Editor of the British Journal of Anaesthesia*

#### UNIFORMITY IN APPARATUS

Dear Sir,—Our attention has been drawn to your correspondent's letter on the subject of "Uniformity in Apparatus", and we should like to bring to your notice the fact that we have, for some months, been in the habit of using the following coloured rubber tubings for connexions to gas cylinder valves



Black	Nitrous oxide
Green	Carbon dioxide
White	Oxygen
Red	Cyclopropane

This procedure has been effected, as your correspondent suggests, by analogy. We have heard of hospitals using red tubing for oxygen, claiming that they were justified in doing so because oxygen was a dangerous gas. The danger of oxygen has never arisen from the gas itself, but from it being a supporter of combustion where a fire has originated.

Years ago, and even to-day, black and red rubber bags are used for gas and oxygen respectively, so that we are not strictly carrying the argument to its logical conclusion, we should, we suppose, use white bags for oxygen, and black for  $N_2O$ .

We are in entire agreement with your correspondent that some authoritative body should make a ruling on this subject, but would like to point out that, as we have been responsible for supplying 90% of the surgical anæsthetic apparatus in this country, and instructing the American makers to follow our colour scheme for their apparatus sent here, this has been done with the approval of the great majority of our anæsthetists. The writer, therefore, thinks that it will only be a question of those disagreeing with the present procedure to justify their disapproval.

Yours faithfully,

p p A Charles King, Ltd  
A CHARLES KING

